The Effects of Alexithymia and Age on Inhibitory Control

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by

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ABSTRACT
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Alexithymia is a stable personality trait typified by externally oriented thinking and difficulties identifying and describing feelings. It is associated with cognitive-affective deficits such as poorer memory for emotional and neutral information as well as executive dysfunction. Relatedly, aging is accompanied by executive dysfunction and increasing alexithymia. Because executive functions comprise multiple cognitive skills, it is essential to demarcate which are impacted by aging and alexithymia. While age-related deficits in inhibitory control are well established, there is a dearth of literature examining inhibition in alexithymia. Thus, this study aimed to examine the effect of alexithymia on inhibition and to interrogate its potential additive impact to aging effects.

Participants were 538 undergraduate students (age = 18-35) and 201 middle-aged to older adults (age = 48-92). All completed the 20-item Toronto Alexithymia Scale (TAS-20) and go, no-go, and stop-signal tasks. Following removal of participants with missing data or invalid task performance, the final sample included 384 younger and 81 older adults. Separate hierarchical regressions predicting accuracy and reaction time were examined. Post hoc models included TAS-20 subscores. Exploratory moderation and mediation models were also conducted to interrogate shared variance among covariates and predictor variables.

Female sex and greater age predicted slower reaction times across all three tasks. Older age was also associated with less accurate responding to target and inhibition trials on no-go and slower and less accurate inhibition on stop. Alexithymia predicted poorer inhibition on no-go and stop via difficulty identifying feelings (DIF). Mood symptoms neither moderated nor mediated the relationship between DIF and inhibitory control.

These results replicate the age-related tradeoff of speed for accuracy in reaction time and inhibition tasks. They also provide novel evidence for alexithymia deficits in non-emotive inhibitory control. The impact of DIF on both automatic (no-go) and conscious (stop) inhibitory control supports processing theories of alexithymia. In particular, DIF contributed to poorer extrinsically and intrinsically cued response suppression. Thus, top-down and bottom-up information processing may be disrupted in alexithymia. Critically, the alexithymia effects were additive to age effects extending support for alexithymia as a risk factor for cognitive aging.

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Introduction

Alexithymia is a personality trait characterized by difficulty identifying feelings (DIF), difficulty describing feelings (DDF), and externally oriented thinking (EOT) (Bagby, Parker, & Taylor, 1994; Bagby, Taylor, & Parker, 1994; Nemiah, 1977; Sifneos, 1973; Taylor, 2000). People who score highly on alexithymia measures are less able to fantasize or use imaginal capacities (Nemiah, Freyberger, & Sifneos, 1976). They tend to have flat affect and communicate through actions and nonverbal behaviors and are distant in interpersonal relationships (Haviland & Reise, 1996). Those who score highly on alexithymia also have deficits in the automatic and conscious processing of emotions likely due to an inability to perceive and label bodily signals that comprise feeling states (Luminet & Zamariola, 2018; Preece, Becerra, Allan, Robinson, & Dandy, 2017).

Alexithymia, manifest at a clinically significant level, is present in 10-13% of adults (Mattila, Salminen, Nummi, & Joukamaa, 2006; Salminen, Saarijarvi, Aarela, Toikka, & Kauhanen, 1999). It is generally more prevalent in men than women, in older adults, and in those with lower socioeconomic status and fewer years of education (Lane, Sechrest, & Riedel, 1998; Mattila et al., 2006; Salminen et al., 1999). However, similar to other personality traits, it is dimensional and distributed continuously across the population (Keefer, Taylor, Parker, & Bagby, 2019; Mattila et al., 2010; Parker, Keefer, Taylor, & Bagby, 2008; Ryder, Sunohara, Dere, & Chentsova-Dutton, 2018).

Alexithymia as a Cognitive-Affective Skill

High alexithymia reflects poorly developed emotional awareness, which is a cognitive skill that develops during childhood and is defined as a person's capacity to

identify and describe their experienced emotions as well as those of others (Lane, Ahern, Schwartz, & Kaszniak, 1997; Lane & Schwartz, 1987). According to the cognitive-developmental theory of emotional awareness, emotions are initially experienced early in life as physical sensations (Lane & Schwartz, 1987). At the next level, they are experienced as action tendencies (e.g., my stomach is growling, therefore I want to eat). Following the development of symbolic thought, action tendencies resulting from physical sensations can be interpreted explicitly as simple emotions (e.g., hunger). Emotional awareness continues to develop alongside more sophisticated and abstract cognitive processes to the point that complex blends of emotions are understood (e.g., "hangry;" cf. MacCormack & Lindquist, 2018).

Contrasting with typical development of emotional awareness, high alexithymia is typified by difficulty differentiating physical sensations from emotions, a tendency to experience negative emotions physically, and hypersensitivity to unpleasant external stimulation (Eastabrook, Lanteigne, & Hollenstein, 2013; Haviland & Reise, 1996; Kano, Hamaguchi, Itoh, Yanai, & Fukudo, 2007; Papciak, Feuerstein, & Spiegel, 1985). Relatedly, the DIF and DDF facets are associated with an inability to construe the meaning of emotions (Inslegers et al., 2012; Kano & Fukudo, 2013; Lane & Schwartz, 1987; Moriguchi & Komaki, 2013). Ultimately, the cognitive skill of emotional awareness appears to be less well developed in people with high alexithymia (Lane et al., 1997; Luminet & Zamariola, 2018).

Executive Functioning in Alexithymia

Given the range of affective deficits in alexithymia, it is important to better understand potential underlying cognitive mechanisms. For example, difficulties with

emotion processing hamper other cognitive abilities such as memory retrieval for emotional content and for neutral information presented in emotive contexts (Dressaire et al., 2015; Luminet, Vermeulen, Demaret, Taylor, & Bagby, 2006; Meltzer & Nielson, 2010; Vermeulen, Domachowska, & Nielson, 2018; Vermeulen & Luminet, 2009). Executive functions are essential to the identification and interpretation of emotions in alexithymia (Vermeulen et al., 2018). They are the higher-order, goal-directed cognitive operations that subserve self-regulation (Elliott, 2003; Hofmann, Schmeichel, & Baddeley, 2012). Many skills comprise executive functioning (EF). They include: working memory (i.e., the capacity to update and manipulate information), set-shifting (i.e., the skill needed to flexibly switch cognitive resources between tasks or mental sets), and inhibitory control (i.e., the ability to suppress irrelevant or interfering stimuli) (Miyake et al., 2000). Importantly, EF impacts the utilization and effectiveness of other cognitive processes, such as self-control and emotion regulation (Hofmann et al., 2012; Zelazo & Cunningham, 2007). As such, executive dysfunction may relate to characteristics of alexithymia (Correro II, Paitel, Byers, & Nielson, 2019; Vermeulen et al., 2018).

Thus far, the literature on EF and alexithymia is small and has limitations. Of the few studies available, some examined the general population while others examined alexithymia induced by neurological injury known as organic alexithymia (e.g., Henry, Phillips, Crawford, Theodorou, & Summers, 2006; Wood & Williams, 2007). The studies of organic alexithymia provide preliminary support for neuropsychological deficits contributing to the cognitive and emotional difficulties present in alexithymia. In one study, adults who had sustained a traumatic brain injury (TBI) were compared with

control participants and showed poorer verbal fluency (Henry et al., 2006). Verbal fluency tasks measure semantic access, search, and control (Strauss, Sherman, Spreen, & Spreen, 2006). Additively, alternating fluency tasks also provide a measure of set-shifting as the participant must switch between semantic categories. Importantly, poorer performance on both semantic and alternating fluency tasks was associated with alexithymia via DIF in the TBI patients and the healthy controls, suggesting a neurological link between cognitive and emotional dysfunction (Henry et al., 2006). Given that the DIF facet isolates poor verbal symbolization of emotions, these findings suggest that difficulty in the rapid generation of words and switching between semantic probes may contribute to elevated DIF.

In another organic alexithymia study, TBI patients were compared with orthopedic patients on measures of alexithymia and a comprehensive neuropsychological battery (Wood & Williams, 2007). The tests were grouped by underlying cognitive domains with sequencing, which reflects the updating executive function proposed by Miyake et al. (2000), and executive ability considered as separate constructs. The TBI group reported greater alexithymia, consistent with emotional changes related to brain trauma, and their alexithymia was related to poorer performance on measures of verbal intelligence and sequencing. No differences arose in the other cognitive domains. However, nontraditional measures comprised the executive ability domain, and the sequencing tasks indexed a subcomponent of EF. Thus, differences between these findings and those of Henry et al. (2006) are difficult to resolve. Although organic alexithymia resulting from TBI may impact some aspects of EF but not others, strong conclusions are premature.

The few studies that have examined alexithymia and EF in the general population primarily support the results stemming from studies on organic alexithymia. A study using only self-report measures in undergraduate students examined emotional intelligence, emotional awareness, alexithymia, and executive functioning (Koven & Thomas, 2010). It linked lower emotional clarity (i.e., DIF and DDF) with more dysfunction across multiple executive skills. An experimental study assessed EF in alexithymia with the Wisconsin Card Sorting Test (Zhu, Wang, Huang, Yao, & Tang, 2006). This test measures problem solving, set-shifting, and conceptual reasoning (Berg, 1948; Grant & Berg, 1948). Examinees are instructed to match cards to one of four key cards with no explicit sorting criteria provided. Feedback from the examiner must be used to discern the sorting strategy. The sorting principle changes after several successful sorts, but the examinee is not explicitly informed of the shift. As such, individuals with deficits in EF may perseverate to a sorting principle after it has changed. They may also fail to maintain a cognitive set, whereby they cannot suppress responding to a different, incorrect sorting strategy. Ultimately, participants with high alexithymia had more total errors, greater perseverative errors, and more failures to maintain set than their low alexithymia counterparts, suggesting poorer problem solving, response inhibition, and set-shifting (Zhu et al., 2006). This limited work extends the organic alexithymia literature by demonstrating executive deficits are present in alexithymia outside the presence of neurological injury.

Inhibitory Control in Later Adulthood

Executive deficits are also present in late adulthood, and losses in EF may underlie age-related changes in cognition and emotion (Goh & Park, 2009; Mather, 2012;

Park et al., 1996). Changes in inhibition may be of central importance (e.g., Hasher & Zacks, 1988). Inhibitory control is the executive function employed in suppressing irrelevant information, removing distracting stimuli from attention, and restraining responses related to irrelevant or distracting information (Lustig, Hasher, & Zacks, 2007). It is an essential component of information processing. Whereas attentional control focuses cognitive processing onto a chosen operation, inhibitory control limits interference from irrelevant information (i.e., access). Impaired access contributes to significant distraction (Darowski, Helder, Zacks, Hasher, & Hambrick, 2008). Another role of inhibition is to remove distracting information once it has entered attentional awareness (i.e., deletion). Without this component, irrelevant content would not be removed from attention or working memory and would therefore compete with more salient stimuli (Charlot & Feyereisen, 2004). Finally, response suppression, or restraint, occurs when an inappropriate or incorrect response is impeded. When response suppression is impaired, an individual presents as impulsive and dysregulated (Hofmann et al., 2012). In late adulthood, an inability to parse irrelevant stimuli from the environment means that more information reaches attentional awareness and thereby burdens working memory resources (Hamm & Hasher, 1992). Furthermore, distracting information cannot be as readily ignored, and it interferes with learning, leading to intrusion errors and false recollections (Hasher & Zacks, 1988). Thus, inhibitory deficits resulting from age may mediate declines in other cognitive skills such as working memory, encoding, and memory retrieval.

While cognitive abilities primarily decline during older adulthood, late-life changes in emotional processes are characterized by both gains and losses. Again,

executive functions, especially inhibition, are central. Older adults experience negative emotions less frequently due, in part, to suppression of negative emotions (i.e., restraint), greater focus on positive information (i.e., access), and less attention toward negative stimuli (i.e., deletion) (Carstensen & Mikels, 2005; Charles, Mather, & Carstensen, 2003; Mather, 2012; Mather & Carstensen, 2005; Mather & Knight, 2005). Despite the declines in EF that accompany aging, older adults frequently recruit executive abilities when presented with emotional stimuli, which allows them to inhibit negative information and focus on positive content (Mather & Knight, 2005). Thus, compensatory recruitment and different emotional goals between older and younger adults may explain the affective changes in older adulthood although this theory remains contentious (Mather, 2012).

Structural changes to the frontal cortex are particularly impactful to inhibitory control, which has been shown to be primarily processed by the prefrontal cortex (Nigg, 2000). For example, increased perseveration that occurs with advancing age is associated with atrophy in the prefrontal cortex (Head, Kennedy, Rodrigue, & Raz, 2009). The anterior cingulate cortex (ACC), within the frontal lobes, is involved in inhibition and is susceptible to age-related alterations (Nigg, 2000; Vaidya, Paradiso, Boles Ponto, McCormick, & Robinson, 2007). Specifically, the dorsal region of the ACC is involved in non-emotional cognitive control, and it is associated with reduced cerebral blood flow and declines in cortical thickness as a function of aging (Egner, Etkin, Gale, & Hirsch, 2008; Vaidya et al., 2007; Whalen et al., 1998). However, ventral regions of the ACC and the prefrontal cortex, which are more predominantly involved in emotional processing, are not as susceptible to cortical thinning in aging (Egner et al., 2008; Fjell et al., 2009; Whalen et al., 1998). The dissociable structural changes between ventral and dorsal

regions support evidence of the preservation of affective processes and degradation of executive abilities, respectively, in older adulthood (Mather, 2012).

Alexithymia and Cognitive Aging

Alexithymia is of central importance to the cognitive, affective, and neural changes associated with aging. It increases with age (Gunzelmann, Kupfer, & Brahler, 2002; Mattila et al., 2006; Paradiso, Vaidya, McCormick, Jones, & Robinson, 2008; Pasini, Delle Chiaie, Seripa, & Ciani, 1992; Salminen et al., 1999), and cognitive difficulties in alexithymia are apparent in adults across the lifespan (Correro II et al., 2019; Dressaire et al., 2015; Lamberty & Holt, 1995; Onor, Trevisiol, Spano, Aguglia, & Paradiso, 2010; Santorelli & Ready, 2015). For example, among healthy adults aged 24-79, greater alexithymia was associated with older age and reduced gray matter volume in the right rostral ACC, suggesting a neuroanatomical substrate for increased alexithymia with older age (Paradiso et al., 2008). Alexithymia was also related to poorer phonemic fluency performance, but age and ACC volume did not covary with phonemic fluency, perhaps due to the small sample and limited range in cognitive abilities. Another study found no age association with alexithymia, but poorer verbal fluency predicted higher alexithymia, particularly high DDF, in older adults and in the total sample of younger and older adults (Santorelli & Ready, 2015).

Recently, a series of three large and non-overlapping experiments demonstrated the contribution of alexithymia to poorer memory and EF across the lifespan (Correro II et al., 2019). Specifically, in a sample of young adults (Experiment 1), EOT predicted poorer delayed memory. In Experiment 2, DIF was negatively associated with performance on EF tasks among young and older adults, and this association was

especially strong when examining the older adults in isolation. Last, Experiment 3 replicated the findings that greater DIF predicted poorer EF and that EOT was associated with poorer delayed memory. This experiment further revealed that among just the older adults, EOT predicted poorer immediate and delayed memory. Additionally, memory was especially poor among older adults with poorer EF, specifically those with higher EOT. Thus, although this literature is quite small, several studies suggest that alexithymia is associated with age and executive deficits, especially in older adults, which may be related to reduced gray matter volume in the right rostral ACC or other frontal circuits (Paradiso et al., 2008; Santorelli & Ready, 2015).

Alexithymia and Inhibitory Control

Inhibitory control is one aspect of EF that should receive more attention in the alexithymia literature given evidence of cognitive biases toward external stimuli (i.e., EOT), attentional capture by somatic sensations (Barsky, Goodson, Lane, & Cleary, 1988; Kano et al., 2007), and difficulties limiting distracting information (Zhang et al., 2011). Regarding the latter, a flanker-type task showed that alexithymia did not impact basic attentional processes such as alerting or orienting, but it prolonged response times and reduced accuracy when deciding whether one arrow in an array of other arrows was pointing in the same or opposite direction (Zhang et al., 2011). This is a measure of conflict processing and requires inhibition of irrelevant stimuli around the target arrow in order to facilitate attention on the target (Eriksen & Eriksen, 1974). Because alexithymia prolonged responding and decreased accuracy, the access and deletion functions of inhibition may be impaired in high alexithymia. Importantly, this study employed neutral

stimuli, indicating alexithymia is associated with generalized inefficiency in attentional control.

Response inhibition has only been directly examined in alexithymia with a task embedded in emotive contexts (Zhang et al., 2012). Specifically, positive, negative, or neutral pictures were superimposed by the letter "M" or "W," and subjects were instructed to respond to "M" but not "W". The task was a modified (i.e., emotive) go/no-go paradigm, which indexes motoric suppression (i.e., the ability to withhold a motor response; Congdon et al., 2012). No significant differences emerged regarding alexithymia in response speed or accuracy, but larger neural responses (P300 event-related potentials), localized to the ACC, were attributable to alexithymia (Zhang et al., 2012). While the alexithymia subscores were not demarcated, it was proposed that DIF could explain the findings such that more DIF leads to less interference from negative emotional contexts. Although the sample was small, it suggested that poorer processing of negative contexts contributes to stronger neural inhibition in high alexithymia. No-go differences due to alexithymia may be more apparent in larger samples when a greater range of alexithymia scores is available. Ultimately, the study's design precluded examination of alexithymia on inhibitory control in a purely neutral task, leaving important questions raised by earlier findings (Zhang et al., 2011) yet to be investigated.

Study Aims

Executive dysfunction is present in both older adulthood and high alexithymia (Onor et al., 2010; Santorelli & Ready, 2015; Vermeulen et al., 2018), and theoretical models of executive deficits in aging and alexithymia implicate alterations in frontal lobe functioning and prefrontal circuitry (Koven & Thomas, 2010; Nielson, Langenecker, &

Garavan, 2002). Moreover, aging is associated with increased alexithymia (Mattila et al., 2006). As such, it is paramount to delineate the specific and unique contributions of alexithymia and age to EF. Because EF comprises a heterogeneous set of separate yet interrelated skills (Miyake et al., 2000), specific executive abilities should be isolated to demarcate which aspects of EF are impacted by aging and alexithymia (Koven & Thomas, 2010).

Inhibitory control is necessary to suppress irrelevant or interfering stimuli. Older adults in particular have difficulty inhibiting intrusive thoughts and suppressing irrelevant information when learning new tasks (Hashtroudi, Johnson, & Chrosniak, 1990; Kausler & Hakami, 1982). Alexithymia is typified by deficits in introspection and stimulus-bound behaviors (Nemiah et al., 1976; Wastell & Taylor, 2002), difficulty with verbal fluency tasks (e.g., Paradiso et al., 2008; Santorelli & Ready, 2015), and perseveration on the Wisconsin Card Sorting Test (Zhu et al., 2006). These EF deficits may stem from difficulty suppressing irrelevant information. Yet, the role of alexithymia on inhibitory control is not well understood.

Thus far, only limited work has been done examining alexithymia and EF. Most of this small literature suggests that working memory updating, semantic control, and abstract speeded information processing are poorer in alexithymia (Correro II et al., 2019; Henry et al., 2006; Lamberty & Holt, 1995; Onor et al., 2010; Paradiso et al., 2008; Santorelli & Ready, 2015). Generally, sample sizes of these few studies have been relatively small, and the results have been mixed even across comparable tasks (e.g., Henry et al., 2006; Lamberty & Holt, 1995; Paradiso et al., 2008; Santorelli & Ready, 2015). Additionally, the focus has been on general alexithymia, rather than its subscores,

although emerging evidence suggests that DIF should receive further study as a specific attribute related to EF (Correro II et al., 2019; Vermeulen et al., 2018). Future studies should build on these findings and extend to more nuanced analysis of alexithymia and measures of inhibition.

The few studies on inhibition in alexithymia were conducted with only younger adults, and much of that work employed emotive tasks, which confounds the examination of executive functions with emotional processing (Vermeulen et al., 2018). Non-emotive inhibitory control is important to examine specifically given the dissociable neural substrates involved in neutral versus emotional cognitive control (e.g., Egner et al., 2008; Whalen et al., 1998). Additionally, inclusion of older adults or a larger age range can help to clarify and address the underlying mechanisms of cognitive effects of alexithymia across the age span where both EF and alexithymia are increasingly relevant.

Purpose

The purpose of this study was to isolate the effects of age and alexithymia on non-emotive tasks that index response inhibition and to delineate the three alexithymia factors. To that end, young and older adults completed a self-report measure of alexithymia and experimental inhibitory control tasks, including go, no-go and stop-signal paradigms (Congdon et al., 2012; Logan, Cowan, & Davis, 1984). The go task builds and sustains prepotent responding to target stimuli, while the no-go task requires selectively responding to the same targets used in the go task (i.e., alternating responding and withholding responses) (Langenecker, Zubieta, Young, Akil, & Nielson, 2007; Nielson et al., 2002). Thus, no-go requires monitoring to switch cognitive sets and inhibit motor responses, and with practice, participants can develop automaticity (Verbruggen &

Logan, 2008). The stop-signal paradigm also measures inhibition, but the inhibitory process is triggered by an external cue (i.e., the "stop" signal) (Logan & Cowan, 1984). Thus, it is more difficult to predict when a response should be inhibited. As such, stop-signal tasks require more controlled and effortful response inhibition (Votruba et al., 2008). By including paradigms that distinguish between automatic and controlled inhibition, this study sought to elucidate the extent to which top-down and bottom-up processing may be impacted in alexithymia.

Hypotheses

The following hypotheses were posed:

1. Age effects:
    a. Relative declines in processing speed and inhibitory control are associated with aging (Nielson et al., 2002), and as such, older adults are expected to be slower when responding to target stimuli.
    b. Relatedly, older adults are expected to be less successful at inhibiting motoric responses during no-go and stop-signal paradigms.
2. Alexithymia effects:
    a. Previous studies have found no difference in high versus low alexithymia with regards to reaction time and the attentional systems of alerting and orienting (Zhang et al., 2012; Zhang et al., 2011). Therefore, high alexithymia is not expected to impact simple processing speed or go task performance.

b. However, conflict monitoring is poorer in high alexithymia (Zhang et al., 2011), and alexithymia is associated with poorer performance on speeded executive tasks (Correro II et al., 2019). These results suggest that accuracy on both the no-go and stop-signal paradigms will be lower in high alexithymia (Zhang et al., 2011). Further, based on self-report measures of working memory, inhibition, and monitoring (Koven & Thomas, 2010) and greater perseverative errors on a conceptual reasoning task (Zhu et al., 2006), alexithymia will be associated with fewer successful inhibition trials for both no-go and stop tasks.

c. Finally, high alexithymia is likely to be associated with slower reaction times to stop signals, indicating more difficulty with executive control (e.g., Zhang et al., 2011).

3. Additive effects:

   a. Some findings suggest executive deficits in high alexithymia are more prominent for older adults (e.g., Correro II et al., 2019; Santorelli & Ready, 2015). Studies with larger sample sizes should have sufficient statistical power to reveal executive dysfunction as a fundamental deficit of alexithymia in addition to age-related difficulties with EF. As such, while alexithymia is expected to be associated with greater age, both age and alexithymia are hypothesized to independently predict EF difficulties. Thus,

alexithymia deficits will be additive to the effects of aging (Dressaire et al., 2015).

4. Effects of alexithymia subscores:
    a. DIF is the salient facet of alexithymia expected to underlie poorer inhibitory control consistent with prior studies demonstrating a relationship between DIF and executive dysfunction (Correro II et al., 2019; Henry et al., 2006; Zhang et al., 2012).

Methods

Participants

The current study utilized archival data from multiple studies on cognition, emotion, and aging. Participants were 538 adult undergraduate students (age = 18-35) and 201 older adults (age = 48-92) recruited from the community. All older adults reported their years of formal education to a maximum of 20, reflecting an advanced degree beyond the master's level (e.g., M.D., Ph.D.). For most young adults, who were all current undergraduate students, years of formal education was estimated as $y_{education} = x_{age} - 6$ to a maximum of 16, reflecting near completion of a bachelor's degree (i.e., senior standing). The older adult sample overrepresented people with risk factors for Alzheimer's disease, including genetic risk (n = 43) and family history (n = 69). All older participants, and some young adults, were screened for intact cognition using protocols specific to each study.

Materials

TAS-20. The 20-item Toronto Alexithymia Scale (TAS-20) is a self-report questionnaire consisting of 20 statements rated for agreement on a 5-point Likert scale (1 = strongly disagree, 5 = strongly agree) (Bagby, Parker, et al., 1994; Bagby, Taylor, et al., 1994). Total scores range from 20 to 100. Scores ≥ 61 are deemed clinically significant alexithymia, and scores 52 to 60 are deemed possible alexithymia (Bagby & Taylor, 1997). While the scale was originally designed with these cutoff scores, alexithymia is increasingly interpreted as a dimensional characteristic with a full range of scores examined across normal populations as we did here (Parker et al., 2008). Three

subscale scores are calculated to reflect the three factors of alexithymia: Difficulty Identifying Feelings (DIF), Difficulty Describing Feelings (DDF), and Externally Oriented Thinking (EOT).

The TAS-20 has good-to-excellent internal consistency and adequate test-retest reliability (Bagby, Parker, et al., 1994). Construct validity is reported as strong, suggesting a robust and consistent three-factor structure although EOT tends to be less strongly associated with the latent alexithymia construct than the DIF and DDF factors (Bagby, Parker, et al., 1994; Bagby, Taylor, et al., 1994; Parker, Taylor, & Bagby, 2003; Preece, Becerra, Robinson, & Dandy, 2018; Sekely, Bagby, & Porcelli, 2018).

Experimental tasks. The following tasks are modified versions of traditional go, no-go, and stop-signal paradigms previously used in the authors' laboratory (Hazlett Elverman, 2016; Langenecker et al., 2007). The tasks were conducted via a computer screen and keyboard using letter stimuli (black font against white background) presented one at a time, consecutively, with no interstimulus interval (0 ms). The font and size of the letters were consistent across all trials.

Go task. Go measures response time and sustained attention (Donders, 1969). Participants were instructed to press the spacebar as quickly and accurately as possible when they saw the letters "r" and "s." This go task was designed to build prepotency for responding to "r" and "s" in the subsequent no-go and stop-signal tasks, which facilitates the examination of response suppression.

No-go task. No-go measures working memory and inhibitory control (Donders, 1969). An extension of the go task, the no-go task required participants to press the spacebar to target letters "r" and "s" but only when they alternated. In other words, they

should not have responded to the same target twice in a row (Langenecker et al., 2007). This task requires selective execution of a response (i.e., intrinsic control), which primarily indexes automatic response inhibition (Rubia et al., 2001; Votruba et al., 2008).

Stop-signal task. Similar to the go and no-go tasks, participants responded to "r" and "s" target letters, but they were instructed to withhold responding if a stop-signal (i.e., a red box) appeared shortly *after* the target. These targets were rare and quasi-random, thereby requiring participants to briefly delay responding to discern whether the stop-signal would occur. The stop-signal delay (SSD) occurred 125-400 ms after stimulus onset, depending on the dataset, but in each study, the delay was varied to prevent prediction. While this procedure is still used, it is now more typical to increase or decrease the SSD on every trial, depending on prior success or failure, to produce 50% correct responses to inhibition trials (Logan et al., 1984). This approach produces a particularly robust SSRT measure with task accuracy equated across participants. Instead, we used the older approach as it allows for an adequate SSRT estimate while also allowing task accuracy to vary (Logan & Cowan, 1984). The stop-signal task, in contrast to the no-go task, employs an external inhibition trigger (i.e., extrinsic control), thereby requiring the participant to *retract* a selective response, which requires more effortful (i.e., top-down) control over motoric responding than no-go (Rubia et al., 2001; Verbruggen & Logan, 2008; Votruba et al., 2008).

Task versions. Given the nature of using archival datasets, task parameters varied somewhat across studies resulting in three versions of the task included in the present study (see Figure 1). Most participants (n = 568) were from studies in which Version 1 was utilized. Seventy-six participants were from studies with a shorter version of the

tasks (i.e., Version 2). For both Versions 1 and 2, each letter was shown up to 750 ms (unless the participant responded sooner). Lastly, 95 participants were from studies in which Version 3 was used where the stimulus duration was 600 ms. In Versions 1 and 2 of the stop-signal task, the stop-signal delays (SSD) were 125 ms and 200 ms; Version 3 had three SSDs: 250 ms, 300 ms, and 400 ms.

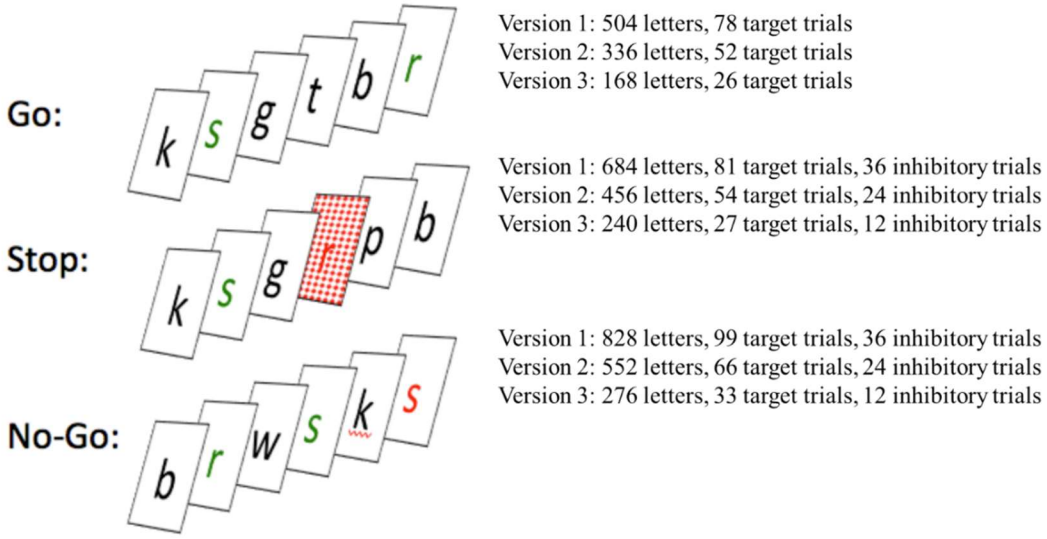

Figure 1. Schematic of the three versions of the experimental tasks. In go, participants respond to the letters "r" and "s." In stop, participants respond to letters "r" and "s" unless a red flash appears (depicted by red checkered above). In no-go, participants respond to "r" and "s" in alternation. Target trials = presentation of the letter "r" or "s" when the participant should respond. Inhibitory trials = presentation of the letter "r" or "s" when the participant should not respond.

MMSE. The Mini-Mental State Examination (MMSE) is a screening instrument for detecting cognitive impairment across five cognitive functions: orientation, verbal learning, attention/calculation, spontaneous verbal recall, and language (Folstein, Folstein, & McHugh, 1975). Scores range from 0 to 30 with scores below 24 suggestive of cognitive impairment although other factors such as education must also be considered when using such cutoffs (Tombaugh & McIntyre, 1992). Many of the older participants

in this study completed the MMSE (n = 199). Those with scores in the normal range (total ≥ 24) were included in the present study. The MMSE has marginal interrater reliability, good test-retest reliability, variable internal consistency, modest to high construct validity, and modest ecological validity (Strauss et al., 2006).

DRS-2. The Mattis Dementia Rating Scale – Second Edition (DRS-2) is a tool used to index the mental status of individuals with known or suspected dementia (Jurica, Leitten, & Mattis, 2001; Mattis, 1988). The instrument measures aspects of attention, initiation, perseveration, visuo-construction, abstract verbal reasoning, and verbal and nonverbal memory. The maximum score for the DRS-2 is 144. An empirically derived cut-off score of 130 was used to demarcate intact cognitive ability for the 103 older participants who completed the task (Monsch et al., 1995). The DRS-2 has good concurrent and predictive validity, and aspects of the instrument have fair reliability and construct validity (Strauss et al., 2006).

NAART. Some older and younger participants (n = 143) completed the North American Adult Reading Test (NAART), which is a word reading task that estimates premorbid intellectual functioning (Blair & Spreen, 1989; Strauss et al., 2006). Participants with scores within normal limits (i.e., scores that are within two standard deviations of the mean of their age group) were included in the study. The NAART has good to excellent reliability and moderate to high construct validity.

Self-report mood measures. Self-report questionnaires were used to quantify symptoms of depression and anxiety to exclude participants with clinically elevated symptomatology and because mood commonly correlates with alexithymia (Honkalampi, De Berardis, Vellante, & Viinamaki, 2018). These measures included the Beck

Depression Inventory—II (BDI-II), Geriatric Depression Scale (GDS), and Beck Anxiety Inventory (Beck & Steer, 1990; Beck, Steer, & Brown, 1996; Yesavage et al., 1982).

The BDI-II evaluates severity of 21 depressive symptoms occurring within the last two weeks (Beck et al., 1996). This instrument has strong psychometric properties including good to excellent internal consistency, high test-retest reliability, and strong convergent and divergent validity (Strauss et al., 2006). Six hundred participants completed the BDI-II.

The GDS is a screening measure of depressive symptomatology for older adults (Yesavage et al., 1982). The long-form (GDS-lf) consists of 30 items whereas the short-form has 15 questions. The short-form (GDS-sf) was completed by 42 participants while the long-form was completed by 48 participants. The long-form has good to excellent internal consistency whereas the short-form appears to have acceptable to good internal consistency (Strauss et al., 2006).

Participants (n = 691) also responded to the BAI, which is an instrument that indexes severity of anxiety symptoms (Beck & Steer, 1990). The BAI has excellent internal consistency and questionable to acceptable test-retest reliability (Beck, Epstein, Brown, & Steer, 1988; Fydrich, Dowdall, & Chambless, 1992). The BAI also has excellent convergent validity, acceptable discriminant validity, and good construct validity (Fydrich et al., 1992).

Mood composite. Raw scores of each of the self-report mood measures were standardized to place them on a normal distribution. A mood composite was calculated for each individual whereby their standardized score on the BAI and their standardized score on either the BDI-II, GDS-sf, or GDS-lf were summed.

Procedure

All procedures were reviewed and approved by Marquette University's Institutional Review Board. The experimental procedures varied based on the larger protocol for each study. For all studies, informed consent was obtained at the beginning of the session. Participants then completed the TAS-20 and the go, no-go, and stop-signal tasks. In Versions 1 and 2 (see Figure 1), participants first completed the go task, then the stop-signal task, and finally, the no-go task; Version 3 reversed the order of no-go and stop. Following the presentation of instructions for each of the task conditions, participants completed blocks of practice trials, except in Version 2.

Sessions involving older adults were conducted individually, and task instructions were read to participants. Sessions with young adults were completed in a group format or individually, but all participants were situated in front of an individual computer and performed the tasks independently.

Data Analytic Plan

A significance criterion of $p < .05$ was used for all statistical tests. All descriptive and inferential statistics were obtained using SPSS v.26. Exploratory mediation and moderation models were conducted using the PROCESS 3.0 custom dialog extension for SPSS (Hayes, 2018). First, participants with missing age, TAS-20, go, no-go, or stop-signal data and duplicated participants were removed from subsequent analyses. Then, participants with impaired scores on the cognitive screening measures were excluded. Next, participants' performances on the go, no-go, and stop-signal tasks were evaluated for poor effort, and dubious responders were removed from subsequent analyses.

Because of the variations between the experimental tasks as well as individual study's procedures, performance metrics were first examined for equivalence across the experimental samples. Then, exploratory correlations with Pearson's *r* for continuous variables and Kendall's *tau* for categorical variables were evaluated to determine which, if any, demographic variables were associated with go, no-go, and stop-signal performance variables. Primary analyses of interest included separate hierarchical regressions predicting accuracy and reaction time for go, no-go, and stop-signal tasks with any significant demographic variables included in initial steps, age entered in the subsequent step, and the TAS-20 total score included in the final step. Post hoc models substituted all TAS-20 subscores (i.e., DIF, DDF, and EOT) for the total score in the final step to explore unique versus shared variance of each factor. Exploratory moderation and mediation models were conducted to interrogate shared variance among covariates and predictor variables.

Dependent variables for accuracy included percent correct target trials (PCTT) for go, no-go, and stop-signal tasks and percent correct inhibitory trials (PCIT) for no-go and stop-signal tasks. Reaction time dependent variables were response time to targets (RTT) for go and stop signal reaction time (SSRT) for stop-signal tasks. SSRT was calculated by finding the probability of incorrectly responding to an inhibitory trial and multiplying that probability by the total number of go response times. The resulting value was used to find the "nth" response time (RT). Each stop-signal delay was subtracted from the "nth" RT. Finally, each of the resultant values was averaged resulting in the SSRT (Logan & Cowan, 1984).

A priori power analyses with a Type I error criterion of .05 and Type II criterion of .80 were conducted using G*Power version 3.1 (Faul, Erdfelder, Lang, & Buchner, 2007) and revealed a total sample size of 98 would be necessary to achieve medium effect sizes for the proposed linear multiple regressions.

Results

Missing and Excluded Data

Participants were excluded from analyses if there were any missing data for the primary predictors (n = 48; 39 younger, 9 older) or the experimental tasks (n = 30; 3 younger, 27 older); task data for one part of the experimental paradigm (e.g., go, no-go or stop) sometimes occurred due to data storage errors, etc. After removing one additional older participant who participated in two of the studies, three participants who had impaired cognitive performance (one younger, two older), and one older participant whose nine years of education were clearly discrepant from all other participants, 656 participants (495 younger, 161 older) were retained for analysis.

To ensure participants completed the go, no-go and stop-signal tasks as instructed and to reduce the likelihood of poor effort, cutoff scores were applied to the PCTT and PCIT metrics. Target performance (PCTT) in each task is fundamental as it demonstrates task engagement and evidence of the prepotent response. If participants did not perform this task with high accuracy, either lack of understanding or insufficient effort were suspected. Previous research using the same tasks as this study demonstrated mean PCTT rates > 90 (Hazlett Elverman, 2016; Langenecker et al., 2007; Votruba & Langenecker, 2013). As such, participants with mean go-PCTT scores < 95 as well as no-go-PCTT and stop-PCTT scores < 90 were excluded from subsequent analyses. Inhibitory control tasks are more challenging relative to simple reaction time measures, and as such, it was important to provide more range for individual differences in task performance. Additionally, the distributions of scores were visually analyzed to approximate cutoffs for

PCIT. Ultimately, a more liberal cut score of 50 was used for mean no-go-PCIT and stop-PCIT scores because it was believed that participants performing above this threshold accurately understood the task instructions and were motivated in performing to the best of their abilities. Thus, subjects were included in analysis if go-PCTT > 95, no-go-PCTT > 90, and stop-PCTT > 90 (i.e., all three filters were applied simultaneously). These cutoff scores excluded 191 participants (111 younger, 80 older), leaving 384 younger and 81 older adults for the final analyses. Final participant demographic characteristics and performance metrics can be found in Table 1.

Table 1
Descriptive Statistics.

	Mean	*SD*	*n*
Demographic Variables			
Age (years)	27.95	19.60	465
Sex (155 M, 310 F)	-	-	465
Education	13.58	1.69	465
Affective Variables			
GDS-lf	2.73	3.18	33
GDS-sf	1.13	1.93	16
BDI-II	7.47	7.11	416
BAI	10.77	8.92	465
TAS-20 Total	43.98	10.41	465
Difficulty Identifying Feelings	12.97	5.11	465
Difficulty Describing Feelings	12.31	4.30	465
Externally Oriented Thinking	18.70	4.29	465
Cognitive and Task Measures			
MMSE	28.99	1.41	81
DRS Total	139.79	2.65	48
NAART	37.21	9.68	82
Go-RTT (ms)	603.56	60.44	464
Go-PCTT	99.67	0.78	464
No-Go-RTT (ms)	660.36	66.88	465
No-Go-PCTT	98.09	2.13	465
No-Go-PCIT	82.70	12.75	465
Stop-RTT (ms)	720.18	78.73	465
Stop-PCTT	98.51	1.98	465
Stop-PCIT	76.30	11.86	465
Stop-SSRT (ms)	488.60	85.30	465

Notes: GDS = Geriatric Depression Scale, -lf = long form, -sf = short form; BDI-II = Beck Depression Inventory - 2nd Edition; BAI = Beck Anxiety Inventory; TAS-20 = Toronto Alexithymia Scale-20; MMSE = Mini-Mental State Examination; DRS = Dementia Rating Scale; NAART = North American Adult Reating Test; RTT = Response Time to Targets; PCTT = Percent Correct Target Trials; PCIT = Percent Correct Inhibitory Trials; SSRT = Stop Signal Reaction Time

Task Differences

Because three versions of the inhibitory tasks were implemented, potential differences between the versions were examined. Importantly, however, this study partly aimed to evaluate age differences on the tasks, and as such, differences between versions due to age were also investigated. Separate one-way ANCOVAs were conducted with task version as the between-subjects variable, age as the covariate, and accuracy metrics or reaction times as dependent variables. All models were nonsignificant (p values for task version ranged from .07 for stop-PCIT to 0.84 for stop-RTT), which suggests that there were no differences in performance metrics based on the version of the task.

Exploratory Correlations

Bivariate correlations are shown in Table 2. Response time to targets across go, no-go, and stop-signal tasks consistently correlated with sex; men responded more quickly than women. Greater mood symptoms were associated with slower response times to targets during go trials, faster response times to targets during stop trials, and poorer stop-signal accuracy. Education was correlated with faster go-RTT and no-go-RTT, slower stop-RTT, and better inhibition (no-go-PCIT and stop-PCIT).

Table 2
Bivariate Correlations.

	Age	Sex	Edu.	Mood	TAS-20	DIF	DDF	EOT	Go-RTT	Go-PCTT	No-Go-RTT	No-Go-PCTT	No-Go-PCIT	Stop-RTT	Stop-PCTT	Stop-PCIT
Age	--															
Sex	0.05	--														
Education	**0.52**	0.04	--													
Mood	**-0.23**	0.07	-0.10	--												
TAS-20	-0.08	-0.03	**-0.12**	**0.47**	--											
DIF	**-0.13**	0.02	-0.09	**0.60**	**0.80**	--										
DDF	**-0.13**	-0.07	**-0.16**	**0.40**	**0.84**	**0.60**	--									
EOT	0.08	-0.01	-0.03	0.02	**0.63**	**0.16**	**0.33**	--								
Go-RTT	**-0.26**	**0.12**	**-0.30**	**0.14**	0.09	0.08	0.10	0.03	--							
Go-PCTT	-0.09	-0.03	0.02	0.04	-0.03	-0.03	0.01	-0.04	0.01	--						
No-Go-RTT	-0.09	**0.11**	**-0.20**	0.08	0.09	0.07	0.09	0.05	**0.81**	<-.01	--					
No-Go-PCTT	**-0.12**	-0.03	-0.02	-0.05	-0.07	-0.06	-0.04	-0.05	-0.01	0.11	-0.08	--				
No-Go-PCIT	0.09	0.05	**0.13**	-0.08	**-0.12**	-0.11	-0.09	-0.07	-0.11	0.09	**-0.25**	**0.36**	--			
Stop-RTT	**0.42**	**0.11**	**0.30**	**-0.14**	-0.08	-0.08	-0.04	-0.04	-0.08	-0.07	0.06	-0.11	0.01	--		
Stop-PCTT	-0.06	-0.05	0.03	-0.09	-0.06	-0.05	-0.03	-0.04	<.01	**0.20**	0.03	**0.30**	**0.17**	-0.06	--	
Stop-PCIT	**0.22**	0.04	**0.26**	**-0.19**	**-0.16**	**-0.19**	**-0.12**	-0.04	**-0.20**	0.02	**-0.14**	0.06	**0.36**	**0.26**	**0.16**	--
Stop-SSRT	**0.19**	0.04	0.06	-0.07	0.01	-0.01	0.04	0.01	0.11	<.01	**0.15**	-0.07	-0.06	**0.63**	0.02	**-0.13**

Notes: <.05; **<.01**; Pearson *r* except with sex (Kendall's τ); Edu. = Education; TAS-20 = Toronto Alexithymia Scale-20; DIF = Difficulty Identifying Feelings; DDF = Difficulty Describing Feelings; EOT = Externally Oriented Thinking; RTT = Response Time to Targets; PCTT = Percent Correct Target Trials; PCIT = Percent Correct Inhibitory Trials; SSRT = Stop Signal Reaction Time.

Hierarchical Regressions

Based on the exploratory correlations, regression models for response time (i.e., go-RTT, no-go-RTT, stop-RTT) included sex in Step 1 to isolate the effect of this variable prior to the predictor variables of interest (i.e., age and alexithymia). Because of the high degree of correlation between education and age (r = .52) and the generally high educational attainment of the sample, this variable was not incorporated into regression models. Mood was incorporated into regression models where it was significantly correlated with primary outcome measures (i.e., go-RTT, stop-RTT, and stop-PCIT; see Table 2). Age was entered in Step 2 of all models, and Step 3 introduced TAS-20 total scores.

Go task. Hierarchical regression produced a significant model for go-RTT. Sex and mood contributed significantly in Step 1 (R^2 = .03, p = .001; see Table 3). Age added significant prediction in Step 2 whereas mood no longer contributed unique variance (R^2 = .086, ΔR^2 = .057, p < .001). Alexithymia did not add substantive prediction in Step 3 although the model remained significant (R^2 = .088, ΔR^2 = .002, p < .001). Thus, older age (β = -.247, p < .001) predicted *faster* go-RTT, and female sex (β = .118, p = .009) was associated with slower go-RTT.

Table 3
Hierarchical Regressions Predicting Go Performances

	Model Summary of Each Step					Contribution of Each Variable in Last Step				
	R^2	ΔR^2	*df*	*F*	*p*	*b*	*SE*	β	*t*	*p*
RTT										
Step 1	0.030	-	2, 461	7.05	**0.001**					
Sex						0.015	0.006	0.118	2.63	**0.009**
Mood						0.002	0.002	0.052	1.00	0.317
Step 2	0.086	0.057	3, 460	14.47	**<.001**					
Age						-0.001	<.001	-0.247	-5.37	**<.001**
Step 3	0.088	0.002	4, 459	11.10	**<.001**					
TAS-20 Total						<.001	<.001	0.051	1.00	0.319
RTT (Older Sample Only)										
Step 1	0.022	-	2, 77	0.85	0.431					
Sex						0.027	0.017	0.114	1.57	0.122
Mood						0.002	0.007	0.022	0.29	0.772
Step 2	0.608	0.586	3, 76	39.31	**<.001**					
Age						0.008	0.001	0.760	10.01	**<.001**
Step 3	0.609	0.001	4, 75	29.19	**<.001**					
TAS-20 Total						<.001	0.001	0.030	0.37	0.710
RTT (Younger Sample Only)										
Step 1	0.034	-	2, 381	6.78	**0.001**					
Sex						0.014	0.004	0.178	3.51	**0.001**
Mood						0.001	0.001	0.053	0.91	0.365
Step 2	0.035	0.001	3, 380	4.59	**0.004**					
Age						0.001	0.003	0.022	0.44	0.660
Step 3	0.037	0.002	4, 379	3.61	**0.008**					
TAS-20 Total						<.001	<.001	-0.047	-0.82	0.414
PCTT										
Step 1	0.008	-	1, 462	3.77	0.053					
Age						-0.004	0.002	-0.093	-2.00	0.047
Step 2	0.009	0.001	2, 461	2.17	0.116					
TAS-20 Total						-0.003	0.003	-0.035	-0.75	0.453

Notes: <.05; **<.01**; RT = Response Time; PCTT = Percent Correct Target Trials; TAS-20 = Toronto Alexithymia Scale-20.

Typically, response times are fastest during young adulthood and gradually slow across middle and later adulthood (Williams, Ponesse, Schachar, Logan, & Tannock, 1999). As such, a post hoc hierarchical regression was calculated separately for older and younger adults to interrogate the atypical association between age and response time. Among older adults, the final model was significant although Step 1 was nonsignificant

with sex and mood entered as predictors (R^2 = .022, p = .431). Age added significant prediction in Step 2 (R^2 = .608, ΔR^2 = .586, p < .001). In Step 3 alexithymia did not contribute unique variance to the model (ΔR^2 = .001). Thus, as expected, older age, but not alexithymia, predicted slower go-RTT (β = .76, p < .001). Conversely, among younger adults, hierarchical regression produced a significant model with sex, but not mood, providing significant prediction in Step 1 (R^2 = .034, p = .001). Neither age in Step 2 nor alexithymia in Step 3 demonstrably altered the model (Step 2 ΔR^2 = .001; Step 3 ΔR^2 = .002). As such, among college-aged adults, only female sex predicted slower go-RTT (β = .178, p = .001).

The models for go-PCTT were nonsignificant. Consistent with the nonsignificant correlation between age and go-PCTT, Step 1 was nonsignificant but approached statistical significance (R^2 = .008, p = .053). Age emerged as a significant predictor in Step 2 when alexithymia was added to the model suggesting that age predicts go-PCTT when the effect of alexithymia is held constant (β = -.093, p = .047). Nevertheless, the model in Step 2 resulted in a reduction of the F value indicating that the initial model better predicted go-PCTT (R^2 = .009, ΔR^2 = .001, p = .116).

No-go task. Hierarchical regression produced a significant model for no-go-RTT (R^2 = .022, p = .018; see Table 4). Sex did not provide unique variance in Step 1 (R^2 = .006, p = .097), age added significant prediction in Step 2 (R^2 = .015, ΔR^2 = .009, p = .034), but none of the individual predictors provided unique variance in Step 3 (ΔR^2 = .007; coefficient ps = .064-.067). Based on this lack of clarity and the post hoc findings for go-RTT, post hoc hierarchical regressions were interrogated separately for the older and younger samples. Among older adults alone, the model was significant (R^2 = .48, p <

.001). Sex did not provide unique variance in Step 1 ($R^2 < .001$, $p = .934$); age added significantly in Step 2 ($R^2 = .479$, $\Delta R^2 = .479$, $p < .001$) and remained significant in Step 3 when alexithymia was added to the model. Alexithymia did not predict no-go-RTT ($\Delta R^2 = .001$). Young adults, in contrast, produced a significant model with only sex demonstrably predicting no-go-RTT across the three steps (final model $R^2 = .03$, $p = .009$). Thus, only age was associated with slower no-go-RTT in older adults ($\beta = .681$, $p < .001$), while only female sex predicted slower no-go-RTT in young adults ($\beta = .172$, $p = .001$).

Table 4
Hierarchical Regressions Predicting No-Go Performances

	Model Summary of Each Step					Contribution of Each Variable in Last Step				
	R^2	ΔR^2	*df*	*F*	*p*	*b*	*SE*	β	*t*	*p*
RTT										
Step 1	0.006	-	1, 463	2.77	0.097					
Sex						0.012	0.007	0.085	1.85	0.065
Step 2	0.015	0.009	2, 462	3.41	0.034					
Age						<.001	<.001	-0.086	-1.85	0.064
Step 3	0.022	0.007	3, 461	3.41	0.018					
TAS-20 Total						0.001	<.001	0.085	1.84	0.067
RTT (Older Sample Only)										
Step 1	<.001	-	1, 79	0.01	0.934					
Sex						0.003	0.024	0.010	0.12	0.907
Step 2	0.479	0.479	2, 78	35.82	**<.001**					
Age						0.008	0.001	0.681	7.91	**<.001**
Step 3	0.480	0.001	3, 77	23.70	**<.001**					
TAS-20 Total						0.001	0.001	0.038	0.44	0.662
RTT (Younger Sample Only)										
Step 1	0.030	-	1, 382	11.64	**0.001**					
Sex						0.015	0.004	0.172	3.40	**0.001**
Step 2	0.030	<.001	2, 381	5.85	**0.003**					
Age						0.003	0.001	-0.015	-0.29	0.772
Step 3	0.030	<.001	3, 380	3.89	**0.009**					
TAS-20 Total						<-.001	<.001	-0.001	-0.03	0.979
PCTT										
Step 1	0.015	-	1, 463	7.12	**0.008**					
Age						-0.014	0.005	-0.13	-2.80	**0.005**
Step 2	0.021	0.006	2, 462	4.93	**0.008**					
TAS-20 Total						-0.016	0.009	-0.08	-1.65	0.100
PCIT										
Step 1	0.008	-	1, 463	3.66	0.056					
Age						0.051	0.030	0.079	1.71	0.089
Step 2	0.021	0.014	2, 462	5.04	**0.007**					
TAS-20 Total						-0.143	0.057	-0.117	-2.53	0.012

Notes: <.05; **<.01**; RT = Response Time; PCTT = Percent Correct Target Trials; PCIT = Percent Correct Inhibitory Trials; TAS-20 = Toronto Alexithymia Scale-20.

The hierarchical regression model predicting no-go-PCTT was significant (R^2 = .021, p = .008). Age contributed significant prediction in Step 1 (R^2 = .015, p = .008), but alexithymia did not add significant prediction in Step 2 (ΔR^2 = .006, p = .008). Thus, only age was associated with poorer no-go-PCTT (β = -.13, p = .005). There was also a

significant model for no-go-PCIT (R^2 = .021, p = .007). Age did not contribute significant prediction in Step 1 (R^2 = .008, p = .056), but alexithymia was associated with poorer no-go-PCIT (ΔR^2 = .014; β = -.117, p = .012).

Stop-signal task. Hierarchical regression produced a significant model predicting stop-RTT with both sex and mood significant in Step 1 (R^2 = .034, p < .001), and only sex and age significant in Steps 2 and 3 (R^2 = .183, ΔR^2 = .149, p < .001 and R^2 = .183, ΔR^2 < .001, p < .001, respectively). Alexithymia was not a significant predictor. Thus, older age and female sex predicted slower stop-RTT (in order, β = .398, p < .001 and β = .094, p = .027; see Table 5). In contrast, the models for stop-PCTT were nonsignificant (R^2 = .008, p = .162), with neither age in Step 1 (R^2 = .004, p = .186) nor alexithymia in Step 2 contributing significant variance (ΔR^2 = .004; coefficient ps > .152).

Table 5
Hierarchical Regressions Predicting Stop-Signal Performances

	Model Summary of Each Step					Contribution of Each Variable in Last Step				
	R^2	ΔR^2	*df*	*F*	*p*	*b*	*SE*	β	*t*	*p*
RTT										
Step 1	0.034	-	2, 462	8.22	**<.001**					
Sex						0.016	0.007	0.094	2.22	0.027
Mood						-0.002	0.002	-0.044	-0.90	0.371
Step 2	0.183	0.149	3, 461	35.46	**<.001**					
Age						0.002	<.001	0.398	9.16	**<.001**
Step 3	0.183	<.001	4, 460	25.83	**<.001**					
TAS-20 Total						<-.001	<.001	-0.018	-0.38	0.708
PCTT										
Step 1	0.004	-	1, 463	1.75	0.186					
Age						-0.007	0.005	-0.067	-1.44	0.152
Step 2	0.008	0.004	2, 462	1.83	0.162					
TAS-20 Total						-0.012	0.009	-0.064	-1.38	0.169
PCIT										
Step 1	0.037	-	1, 463	17.77	**<.001**					
Mood						-0.710	0.346	-0.106	-2.05	0.040
Step 2	0.070	0.033	2, 462	17.49	**<.001**					
Age						0.115	0.028	0.190	4.13	**<.001**
Step 3	0.077	0.006	3, 461	12.79	**<.001**					
TAS-20 Total						-0.103	0.058	-0.091	-1.79	0.073
PCIT (Remodeled)										
Step 1	0.049	-	1, 463	24.01	**<.001**					
Age						0.127	0.027	0.210	4.67	**<.001**
Step 2	0.068	0.019	2, 462	16.96	**<.001**					
TAS-20 Total						-0.158	0.051	-0.139	-3.08	**0.002**
PCIT (Older Sample Only)										
Step 1	0.146	-	1, 79	13.48	**<.001**					
Age						-0.399	0.117	-0.374	-3.42	**0.001**
Step 2	0.146	0.001	2, 78	6.69	**0.002**					
TAS-20 Total						-0.032	0.139	-0.026	-0.23	0.816
PCIT (Younger Sample Only)										
Step 1	0.032	-	1, 372	12.43	**<.001**					
Age						1.334	0.393	0.170	3.40	**0.001**
Step 2	0.047	0.015	2, 381	9.39	**<.001**					
TAS-20 Total						-0.135	0.054	-0.125	-2.49	0.013
SSRT										
Step 1	0.035	-	1, 463	16.72	**<.001**					
Age						0.001	<.001	0.189	4.12	**<.001**
Step 2	0.036	0.001	2, 462	8.53	**<.001**					
TAS-20 Total						<.001	<.001	0.028	0.61	0.545

Notes: <.05; **<.01**; RT = Response Time; PCTT = Percent Correct Target Trials; PCIT = Percent Correct Inhibitory Trials; SSRT= Stop-Signal Reaction Time; TAS-20 = Toronto Alexithymia Scale-20.

Hierarchical regression analysis revealed a significant model predicting stop-PCIT with mood significant across all three steps (Step 1: $R^2 = .037$, $p < .001$), and age contributing in Step 2 ($R^2 = .07$, $\Delta R^2 = .033$, $p < .001$). When alexithymia was added in Step 3, the effect of mood was reduced ($R^2 = .077$, $\Delta R^2 = .006$, $p < .001$; $\beta = -.149$, $p = .001$ in Step 2, $\beta = -.106$, $p = .04$ in Step 3), while the effect of age was unchanged ($\beta = .188$, $p < .001$ in Steps 2 and $\beta = .19$, $p < .001$ in Step 3). Moreover, alexithymia approached statistical significance ($\beta = -.091$, $p = .073$). Partial correlations were examined to further elucidate the relationships among the variables. The zero-order correlation of TAS-20 with stop-PCIT was significant ($r = -.16$, $p = .001$). However, the partial correlation between these variables, in which the effect of mood was controlled, was considerably less ($r = -.08$, $p = .098$). That is, the correlation coefficient was diminished by half. As such, the inclusion of mood severely reduced the amount of variance in stop-PCIT shared by alexithymia. Thus, models for stop-PCIT were influenced by multicollinearity. To address this issue, the data were remodeled: age was significant in Step 1 ($R^2 = .049$, $p < .001$), and both age and alexithymia were significant in Step 2 ($R^2 = .068$, $\Delta R^2 = .019$, $p < .001$). Therefore, older age was associated with *better* stop-PCIT ($\beta = .21$, $p < .001$), and greater alexithymia predicted poorer stop-PCIT ($\beta = -.139$, $p = .002$).

Given that greater stop-PCIT accuracy was associated with older age, in contrast to expectations, follow-up models were evaluated separately across age samples. Older adults alone produced a significant model with age predicting poorer PCIT performance, as predicted ($R^2 = .146$, $p < .002$; $\beta = -.382$, $p < .001$), with no added effect of alexithymia in Step 2 ($R^2 = .146$, $\Delta R^2 = .001$, $p = .002$). In contrast, young adults alone also produced

a significant model but with greater age predicting better performance (R^2 = .032, p < .001) and alexithymia adding further prediction in Step 2 (R^2 = .047, ΔR^2 = .015, p < .001). Thus, aging was associated with poorer stop-PCIT, as predicted, but among young adults, stop-PCIT improved with age whereas alexithymia impaired it (β = .17, p = .001 and β = -.125, p = .013, respectively).

Last, hierarchical regression produced a significant model predicting stop-SSRT with age significant in Step 1 (R^2 = .035, p < .001). Alexithymia did not add significant prediction in Step 2 (R^2 = .036, ΔR^2 = .001, p < .001; β = .028, p = .545). Thus, only age was associated with slower stop-SSRT (β = .189, p < .001).

Post Hoc Examination of PCIT Findings

In order to specify the salient subscales of alexithymia accounting for inhibitory control deficits, post hoc hierarchical regression models were performed with the three TAS-20 subscores replacing the total score in Step 3 (see Table 6). Part and partial correlations were computed, and the subscore with the highest part correlation coefficient was targeted for follow-up moderation and mediation analyses.

Table 6
Post Hoc Hierarchical Regressions Predicting PCIT Performances Using Factor Scores

	Model Summary of Each Step					Contribution of Each Variable in Last Step				
	R^2	ΔR^2	*df*	*F*	*p*	*b*	*SE*	β	*t*	*p*
No-Go-PCIT										
Step 1	0.008	-	1, 463	3.66	0.056					
Age						0.053	0.031	0.081	1.73	0.085
Step 2	0.022	0.014	4, 460	2.62	<u>0.035</u>					
DIF						-0.214	0.144	-0.086	-1.49	0.137
DDF						-0.033	0.180	-0.011	-0.18	0.856
EOT						-0.182	0.147	-0.061	-1.24	0.215
Stop-PCIT										
Step 1	0.037	-	1, 463	17.77	**<.001**					
Mood						-0.577	0.383	-0.086	-1.51	0.133
Step 2	0.070	0.033	2, 462	17.49	**<.001**					
Age						0.117	0.028	0.193	4.16	**<.001**
Step 3	0.080	0.009	5, 459	7.93	**<.001**					
DIF						-0.260	0.149	-0.112	-1.74	0.082
DDF						0.041	0.163	0.015	0.25	0.800
EOT						-0.001	0.001	-0.035	-0.71	0.476

Notes: <u>≤.05</u>; **<.01**; RT = Response Time; PCTT = Percent Correct Target Trials; PCIT = Percent Correct Inhibitory Trials; SSRT = Stop Signal Reaction Time; TAS-20 = Toronto Alexithymia Scale-20; DIF = Difficulty Identifying Feelings; DDF = Difficulty Describing Feelings; EOT = Externally Oriented Thinking.

No-go task. Hierarchical regression predicting no-go-PCIT produced a significant model. Age approached significance in Step 1 (R^2 = .008, p < .056; β = .089, p = .056). In Step 2, the model was significant (R^2 = .022, ΔR^2 = .014, p = .035), but none of the predictors (i.e., age, DIF, DDF, EOT) reached the threshold for significance (ps = .085-.856) suggesting issues with multicollinearity. To clarify the model, the partial and semi-partial correlations from Step 2 were examined. After controlling for the effects of the other variables, age accounted for the most variance (r = .08) followed by DIF (r = -.069). Thus, DIF appeared to account for the most variance among the alexithymia factors, and it predicted poorer no-go-PCIT.

Despite substantial research showing alexithymia is a stable personality trait (Taylor & Bagby, 2004), individuals with high depression and anxiety symptoms also tend to have high alexithymia scores leading some to argue that alexithymia is an affective state of psychiatric distress (Honkalampi et al., 2010; Marchesi, Ossola, Tonna, & De Panfilis, 2014). In the present study, the mood composite was moderately-to-strongly correlated with TAS-20 (r = 0.47) and DIF (r = .60). As such, follow-up analyses were evaluated for the possibility of moderation or mediation by the mood composite. First, moderation was not supported; despite a significant overall moderation analysis (R^2 = .023, p = .033), none of the predictors (i.e., DIF, mood, DIF-X-mood interaction) achieved significance (ps = .102-.238). Second, bootstrapped mediation analysis demonstrated that the mood composite did not mediate the relationship of DIF to no-go-PCIT (see Figure 2). Specifically, while mood did reduce the direct effect relative to the total effect of DIF to no-go-PCIT ($b_{c'}$ = -0.243, p = .092 and b_c = -0.256, p = .028, respectively), and alexithymia provided significant prediction of the mood composite (b_a = 0.201, p < .001), the mood composite did not predict no-go-PCIT (b_b = -0.065, p = .878). The lack of mediation was verified by examining the 95% confidence interval of the indirect effect (b_{ab} = -0.013, 95% CI [-0.186, 0.153]). The null hypothesis could not be rejected because the 95% CI included zero. Thus, higher DIF uniquely predicted lower no-go-PCIT, and mood neither moderated nor mediated this relationship.

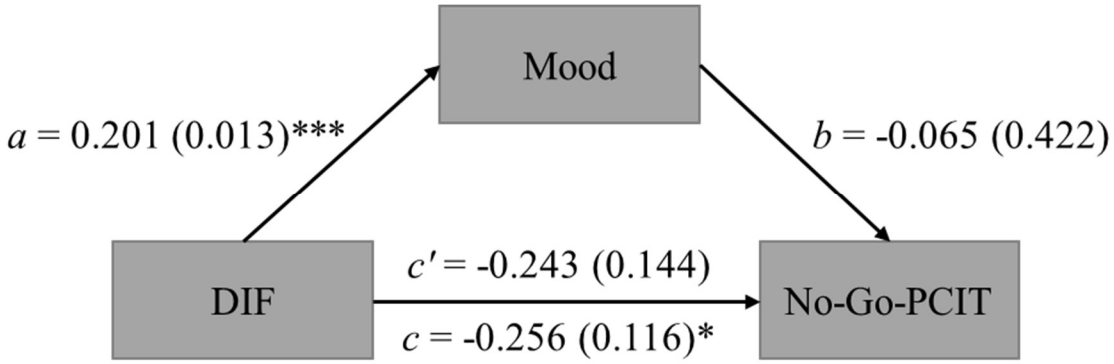

Figure 2. DIF was associated with mood (*a*), but mood was not related to no-go-PCIT (*b*). While mood reduced the direct effect (*c*) relative to the total effect (*c′*), the 95% confidence interval of the indirect effect (*ab*) contained zero (95% CI [-0.186, 0.153]). Thus, mood did not mediate the relationship between DIF and no-go-PCIT. Reported values are coefficients and standard errors. *p <.05, **p<.01, ***p<.001

Stop-signal task. Models predicting stop-PCIT were significant (R^2 = .08, p < .001). Mood provided significant prediction in Step 1 (R^2 = .037, p < .001; β = -.192, p < .001), age added prediction in Step 2 (R^2 = .07, ΔR^2 = .033, p < .001; β = .188, p < .001), and in Step 3, only age remained as a significant predictor (ΔR^2 = .009, p < .001, β_{age} = .193, p < .001). That is, mood was no longer a unique predictor (β = -.086, p = .133), and DIF was marginally significant (β = -.112, p = .082). The part correlation coefficient for age was largest of the predictors (r = .186) followed by DIF (r = -.078) then the mood composite (r = -.07) suggesting that DIF was the most salient facet of alexithymia after controlling for the effect of the other predictors on stop-PCIT.

Because anxiety and depression are often correlated with alexithymia, additional models were examined to discern whether mood moderated or mediated the relationship between DIF and stop-PCIT. First, mood did not moderate the role of DIF on stop-PCIT. Although the model was significant (R^2 = .079, p < .001), none of the predictors were (mood, DIF, mood-X-DIF interaction; ps = .082-.931); only the covariate age was

significant (b = .115, p < .001). Second, bootstrapped mediation analysis revealed that mood did not mediate the effect of DIF on stop-PCIT (see Figure 3). That is, DIF and mood were associated (b_a = 0.201, p < .001), mood did not predict stop-PCIT (b_b = -0.547, p = .151), and DIF predicted stop-PCIT (b_c = -0.369, p = .001). Importantly, the direct effect of mood on stop-PCIT, although reduced, remained significant ($b_{c'}$ = -0.259, p = .046). This was confirmed by examining the 95% confidence interval of the indirect effect (b_{ab} = -0.11, 95% CI [-0.259, 0.033]), which suggested that the indirect effect was not significantly different from zero.

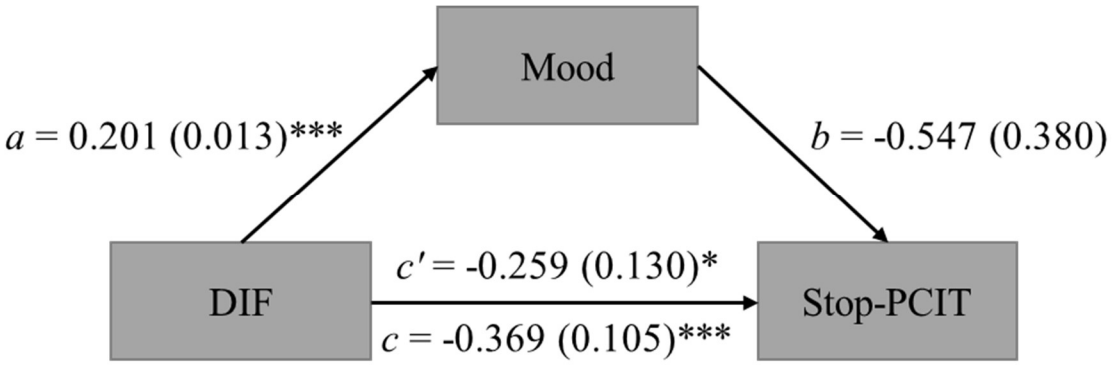

Figure 3. DIF was associated with mood (*a*), but mood was not related to stop-PCIT (*b*). While mood reduced the direct effect (*c*) relative to the total effect (*c′*), the 95% confidence interval of the indirect effect (*ab*) contained zero (95% CI [-0.259, 0.033]). Thus, mood did not mediate the relationship between DIF and stop-PCIT. Reported values are coefficients and standard errors. *p <.05, **p<.01, ***p<.001

Discussion

The purpose of this study was to investigate the effects of age and alexithymia on inhibitory control among adults ranging in age from 18-92 years. Participants completed tasks of motoric inhibition: go, no-go and stop-signal paradigms. Results revealed female sex and older age were associated with slower responding on target trials across go, no-go, and stop-signal tasks. Greater age predicted less accurate responding to target trials on no-go and slowed processing of stop signals. Among the middle-aged to older adults, greater age was associated with less accurate responding to inhibition trials in the stop-signal paradigm. Importantly, greater alexithymia predicted less accurate responding to inhibitory trials on no-go. Difficulty identifying feelings (DIF) was the salient facet of alexithymia that accounted for this effect. Similarly, while anxiety and depression symptoms also predicted poorer performance on inhibitory trials of the stop-signal task, this appeared to be due to shared variance between mood and alexithymia. Again, DIF was the subscale of alexithymia accounting for this effect. Finally, mood neither moderated nor mediated the relationship between DIF and inhibitory control on the no-go and stop-signal tasks. Taken together, this study captured typical aging findings related to slower, but generally accurate, responding on go, no-go and stop-signal paradigms, and alexithymia independently contributed to poorer inhibitory control via DIF.

Age Effects

Hypothesis 1A: Slower RTT with greater age. Prior studies have demonstrated that while older age contributes to slower responding on go and no-go tasks, accuracy remains consistent across older and younger samples (Hazlett Elverman, 2016; Nielson et

al., 2002). In this study, age significantly predicted RTT in go, no-go, and stop-signal tasks, but only its association with stop-RTT was in the predicted direction. This appeared to be driven by the more limited spread of RTTs in the younger sample. When older adults were examined separately, greater age predicted slower go-RTT and no-go-RTT, as was expected. For young adults, age was not a significant predictor of go-RTT and no-go-RTT. Typically, simple reaction times are fastest among young adults with slowing beginning in midlife (Der & Deary, 2006). As many of the young adults completed the tasks in group settings, they may have been distracted or less motivated to perform well (as often occurs with student samples), potentially delaying their RTT. In contrast, the stop-signal task requires more effortful control over responding, which appears to have been better exerted by the young adults under these strong top-down control requirements (Verbruggen & Logan, 2008). Alternatively, there was a very large sample of young adults with a narrow performance distribution, combined with a smaller sample of older adults who had a wider performance distribution. Furthermore, the distribution was bimodal, with a lack of representation of participants age 25-50. A fuller and more balanced age distribution would likely have produced reaction times more representative of prior studies.

Although target accuracy during the no-go and stop tasks were expected to be uninfluenced by age, poorer no-go-PCTT was apparent at older ages. Indeed, normative data reveal a negative trend for no-go-PCTT across adulthood, but this trend typically does not reach statistical significance, especially due to typically high overall task performance (Nielson et al., 2002; Votruba & Langenecker, 2013). The significant effect of age on no-go-PCTT in the present study was small but significant likely due to the

large sample size. Similarly, its magnitude is consistent with prior literature (Nielson et al., 2002). While both go and no-go are relatively easy, target detection in the no-go task is more challenging relative to go because no-go imposes a small working memory requirement in order to accurately alternate responding (Garavan, Ross, & Stein, 1999; Nielson et al., 2002; Wingfield, Stine, Lahar, & Aberdeen, 1988). Yet, such aging effects also likely have little meaning due to the ceiling effect on performance (floor = 95% correct).

Hypothesis 1B: Poorer PCIT and slower SSRT with greater age. In contrast to target detection, greater age was expected to predict fewer correct inhibitory trials on no-go and stop-signal tasks and slower inhibitory control processes (Hazlett Elverman, 2016; Nielson et al., 2002; Rey-Mermet & Gade, 2018). The results partly supported this hypothesis. Although age did not predict no-go-PCIT, older age was significantly associated with poorer stop-PCIT within the older group. Moreover, greater age predicted slower SSRT.

No-go task. The lack of an age effect on no-go-PCIT is inconsistent with a recent meta-analysis (Rey-Mermet & Gade, 2018). Across 18 studies with go and no-go tasks, aging was associated with more errors on inhibition trials. Yet, our sample was highly educated, and we set a high criterion for task performance to assure both adequate understanding of and engagement with the tasks; these factors may not have been considered in other studies. Moreover, our strict filter criterion resulted in a relatively small sample of older adults (n = 81). Using the same cutoff for all participants, regardless of age, likely resulted in the loss of older individuals who understood the

instructions and provided sufficient effort. Thus, the present results may reflect a Type II Error, such that a true aging effect was not captured.

A prior study (some participants overlapped with this study) also did not detect age group differences for inhibitory trials on the no-go task; however, there were significant group differences in event-related potential (ERP) amplitudes during inhibitory control (Hazlett Elverman, 2016). Specifically, older adults had greater frontal (midline) amplitudes and reduced central-parietal (midline) amplitudes during no-go relative to young adults suggesting older adults had less efficient processing in posterior brain regions and neural recruitment in frontal regions, which is consistent with compensatory models of aging (Cabeza, 2002; Nielson et al., 2002; Park & Reuter-Lorenz, 2009; Reuter-Lorenz & Park, 2014). Thus, an alternative explanation for the nonsignificant impact of age on no-go-PCIT is that the participants in the current study were able to effectively compensate for age-related changes.

Stop-signal task. As expected, older age predicted poorer stop-PCIT among the sample of middle-aged to older adults and slower stop-SSRT across all participants. These results support Hypothesis 1B (i.e., poorer inhibitory control with older age). Yet, for college students, age predicted *better* stop-PCIT consistent with the ongoing development of executive functions through young adulthood and into early adulthood (Friedman et al., 2016).

Commensurate with a recent meta-analysis, older age was associated with both less accurate responding to inhibition trials and slowed suppression of motoric responses (Rey-Mermet & Gade, 2018). Our prior work using this stop-signal task and a portion of the same participants found no significant age group difference for stop-PCIT but did

show greater ERP amplitude in older adults (Hazlett Elverman, 2016) indicative of less efficient processing and compensatory recruitment that occurs as a function of aging (Cabeza, 2002). ERP may therefore be a more sensitive method for capturing age-related changes in smaller studies. In larger studies, such as this one, age-related differences in stop-PCIT may be more likely to emerge.

Differential age effects in no-go vs. stop-signal. Task demands may explain the variable aging findings between no-go-PCIT and stop-PCIT. On average, all participants performed more poorly on inhibition trials of the stop-signal task compared to no-go. This finding supports the automatic-inhibition hypothesis, which predicts that as an individual learns the pattern between target and inhibition trials they can anticipate when a response should be inhibited leading to more automatic and accurate inhibitory control (Verbruggen & Logan, 2008). No-go in particular is better suited for the development of automaticity. In the stop-signal paradigm, the stimuli for responding and for inhibiting are the same (Votruba et al., 2008). Therefore, the stop and the go responses are inconsistent and less likely to become automatized reflecting more controlled and effortful response suppression relative to no-go (Verbruggen & Logan, 2008). Yet, for both tasks automatic response inhibition can develop with practice. Nevertheless, for no-go the demand on response retraction is less reliant on top-down control and is more likely to be mediated by implicit learning (Rubia et al., 2001; Verbruggen & Logan, 2008). Thus, motoric suppression via the stop-signal task may be better suited to ascertain age-related changes across middle to late adulthood.

Alexithymia Effects

An important objective of this study was to examine potential independent effects for both age and alexithymia on inhibitory control. The hierarchical regressions were designed to isolate age effects after accounting for significant covariates and then to distinguish the extent to which alexithymia effects were additive to aging (cf., Dressaire et al., 2015). This study also sought to demarcate which of the facets of alexithymia contributed to inhibitory control deficits.

Hypothesis 2A: Alexithymia should not affect responding to target trials. In prior studies, alexithymia did not impact simple processing speed and response accuracy in non-emotive or emotive inhibition tasks (Zhang et al., 2012; Zhang et al., 2011). Thus, no alexithymia effects were anticipated for go-PCTT, go-RTT, no-go-PCTT, no-go-RTT, stop-PCTT, or stop-RTT. The results supported the hypothesis (see Tables 3-5).

Hypotheses 2B, 2C, and 4A: Poorer inhibition with greater alexithymia via DIF. Previous studies revealed that high alexithymia is associated with less accurate and slower conflict processing (Zhang et al., 2011); more perseverative errors on a conceptual reasoning task (Zhu et al., 2006); greater self-reported difficulties with working memory, inhibition, and monitoring (Koven & Thomas, 2010); and deficits on traditional executive functioning tasks (Correro II et al., 2019; Santorelli & Ready, 2015; Wood & Williams, 2007). As such, alexithymia was hypothesized to independently contribute to poorer inhibitory control as measured by no-go-PCIT, stop-PCIT, and stop-SSRT.

No-go task. As expected, alexithymia predicted poorer no-go-PCIT. Post hoc analyses with the three facets of the TAS-20 were opaque due to shared variance of the subscales (see Table 2; see also Preece et al., 2018), but DIF had the largest partial and

semi-partial correlations after age. Further analysis showed that DIF predicted no-go inhibitory performance and that it did so independent of mood (with which it is highly correlated). These results support previous demonstrations of executive deficits in alexithymia via DIF (Correro II et al., 2019; Henry et al., 2006; Zhang et al., 2012).

Stop-signal task. A complicated relationship emerged for the stop-signal performance metrics. First, neither alexithymia nor any of its subscales predicted SSRT. Second, mood predicted poorer stop-signal inhibition while neither the total score of the TAS-20 nor any of its salient subscores uniquely predicted stop-PCIT. However, inclusion of alexithymia in the model reduced or eliminated the mood effect. As alexithymia and mood (i.e., anxiety and depression) are highly correlated (Honkalampi et al., 2018), this finding suggested a shared variance issue. As previous studies reveal small to no effects of anxiety and depression on inhibitory control tasks, it seemed likely that alexithymia was driving the stop-PCIT results (Lipszyc & Schachar, 2010; Lyche, Jonassen, Stiles, Ulleberg, & Landro, 2010; Wright, Lipszyc, Dupuis, Thayapararajah, & Schachar, 2014). Age again had the largest partial and part correlations, but DIF was more strongly associated with stop-PCIT than mood and was independent of possible moderation and mediation effects of mood despite the high correlation between these constructs. Thus, greater alexithymia, via DIF, predicted poorer inhibition accuracy, which extends earlier studies demonstrating the importance of DIF on executive functioning (Correro II et al., 2019; Henry et al., 2006; Zhang et al., 2012). The findings also substantiate the argument that alexithymia is related to, but not attributable to, anxiety and depression (Honkalampi et al., 2018). Finally, the effect of alexithymia on inhibitory performance, but a lack of effect on SSRT, was consistent with prior work

showing alexithymia does not contribute to simple processing speed but is associated with both slower and less accurate conflict processing (Zhang et al., 2011). Importantly, these errors may underpin other emotional-behavioral response difficulties in alexithymia (Taylor & Bagby, 2004).

Interpretations and interim summary. Emerging evidence suggests a general cognitive deficit in high alexithymia, and the present results support the notion that alexithymia contributes to impairments in executive functions. Prior studies that did not differentiate the TAS-20 subscales demonstrated elevated self-report of executive deficits, greater errors on sequencing tasks, and impaired conflict processing in high alexithymia (Koven & Thomas, 2010; Wood & Williams, 2007; Zhang et al., 2011). When TAS-20 factors have been demarcated, DIF, and less frequently DDF, accounted for executive dysfunction (Correro II et al., 2019; Henry et al., 2006; Santorelli & Ready, 2015). As such, impaired executive functioning may underlie the emotion processing difficulties in alexithymia, and deficient emotion processing may be one mechanism through which people with high alexithymia also have high psychiatric distress (Correro II et al., 2019; Honkalampi et al., 2018; Li, Zhang, Guo, & Zhang, 2015; Lumley, 2000; Marchesi, Brusamonti, & Maggini, 2000; Parker, Bagby, & Taylor, 1991). Only one prior study examined inhibitory control utilizing go and no-go tasks, reporting no alexithymia differences in task performance (Zhang et al., 2012). Importantly, the sample was small (n = 30) and the experimental paradigm was conducted in an emotional context thereby limiting interpretations about neutral response inhibition. Regardless, that study did reveal neural activation differences in inhibitory control for negative contexts that were

attributable to alexithymia; although not directly assessed, DIF was proposed as the facet likely responsible.

The present results demonstrated a negative association between alexithymia and inhibitory control performances consistent with the characterization of alexithymia as a deficit in the transfer of information from subsymbolic physiological sensations to symbolic thought necessary for acting on and regulating behaviors (Bucci, 2001; Frawley & Smith, 2001; Lane et al., 1997; Luminet & Zamariola, 2018; Murphy, Catmur, & Bird, 2017; Preece et al., 2017; Rinaldi, Radian, Rossignol, Arachchige, & Lefebvre, 2017; Vermeulen et al., 2018). We provide evidence of poorer bottom-up and top-down control over response inhibition in alexithymia that sheds light on the perspective of alexithymia deficits in stimulus appraisal, action preparedness, and execution. Importantly, the inhibition tasks were non-emotive suggesting that an information processing deficit in alexithymia extends to neutral contexts. This supports an emerging body of literature indicative of general cognitive deficits in alexithymia (Correro II et al., 2019; Vermeulen et al., 2018).

DIF is uniquely situated at the intersection of bottom-up emotional awareness and top-down interpretation of social and emotional information (Bar-On, Tranel, Denburg, & Bechara, 2003; Frawley & Smith, 2001), which may make both selective response execution and extrinsic response retraction challenging. The response inhibition tasks used in the current study are capable of measuring both bottom-up and top-down inhibitory processes (Verbruggen & Logan, 2008). DIF contributed to poorer automatic response inhibition (i.e., no-go-PCIT) and controlled response inhibition (i.e., stop-PCIT). From a processing framework, an inability to ascertain the meaning of stimuli in

the environment and within the self leads to an inability to plan or prepare an action response (Frawley & Smith, 2001). This disconnection might explain the elevated commission errors on a stop-signal task. Namely, DIF impedes one's capacity to integrate extrinsic information properly and thereby execute an appropriate response, which could lead to elevated commission errors to lures. Relatedly, a disconnect between action readiness and execution could explain the no-go results (Frawley & Smith, 2001). That is, participants knew that they should alternate between inhibiting and responding to two target stimuli; however, DIF interfered with the appropriate and flexible execution of this behavior. These results are consistent with the inappropriate implementation of emotional behaviors, such as incongruent or flat affect, present in high alexithymia (Taylor & Bagby, 2004).

Ultimately, an inability to interpret one's emotions contributed to poorer control over motoric responses. Stated differently, impairments in automatically and consciously controlling goal-directed behavior may impute difficulty in interpreting one's sensations as emotional phenomena. The proposed deficit in both bottom-up and top-down informational transfer appears to happen at the rate of milliseconds potentially explaining the relatively small effect sizes in the present work. Because of the rapidity of these cognitive processes, both conscious and preconscious, more sensitive methods might assist in understanding informational transfer in high alexithymia. Indeed, recent electrophysiological work has revealed deficits in both early, automatic and later, conscious processing of affective information (Goerlich, 2018). While many anatomical structures have been implicated in high alexithymia, the anterior cingulate cortex (ACC), a structure that is critical for controlling emotions and behaviors, including motoric

responding, may be essential for understanding the link between the emotive and non-emotive cognitive deficits present in high alexithymia (Goerlich & Aleman, 2018; Lane et al., 1997; Zhang et al., 2012). Future studies are needed to better ascertain the role of the ACC and early attentional processes for non-emotive stimuli in alexithymia.

Age and Alexithymia Effects

Hypothesis 3A: Alexithymia effects additive to age effects. Age and alexithymia independently predicted poorer performance on two tasks of inhibitory control, as predicted. These results highlight the importance of alexithymia as a risk factor for poorer cognitive functioning across the lifespan and especially in older adulthood. Aging is typified by declines in cognitive processes, including executive functions such as inhibitory control (Caserta et al., 2009; Hasher & Zacks, 1988; Salthouse, 2010). Alexithymia is also associated with deficits in executive processes and, in this study, response inhibition (Correro II et al., 2019; Vermeulen et al., 2018). Specifically, alexithymia via DIF added significant predictive value beyond aging effects on measures of inhibition. As such, the effect of alexithymia on cognition may be particularly impactful during late life.

Although this study was not designed to directly examine neural functioning, the present results may be useful in understanding the role of the ACC in aging, inhibition, and alexithymia. The dorsal region of the ACC is involved in neutral cognitive control (including inhibitory control), is associated with age-related functional and structural declines, and appears to be reduced in high alexithymia (Egner et al., 2008; Goerlich & Aleman, 2018; Vaidya et al., 2007; Whalen et al., 1998). In this study, high alexithymia

was associated with behavioral deficits on inhibitory control tasks. Given the role of the dorsal ACC in inhibition and the relative reductions of dorsal ACC volume in high alexithymia (Goerlich & Aleman, 2018; Nigg, 2000), the present results may reflect the neurobehavioral consequences of aberrant neuroanatomical structure and function present in high alexithymia. Moreover, the dorsal ACC is susceptible to cortical thinning in aging and potentially provides a neural mechanism for alexithymia as a risk factor for cognitive aging (Egner et al., 2008; Fjell et al., 2009; Whalen et al., 1998). In fact, some studies claim that alexithymia reflects a generalized neurocognitive functioning deficit, is a neuropsychiatric consequence of normal aging, and uniquely predicts pathological cognitive aging (Messina, Beadle, & Paradiso, 2014; Paradiso et al., 2008; Ricciardi, Demartini, Fotopoulou, & Edwards, 2015; Sturm & Levenson, 2011; Yuruyen et al., 2017). Additional research will be necessary to parse out the complex interplay of cognition, age, and alexithymia.

Limitations

The studies combined for this project attempted to provide a relatively full age spectrum. Despite this intention, age was still bimodally represented with an inordinate number of very young adults (i.e., under 25 years; college samples) and lacking most of the middle age spectrum. As such, it was necessary in some occasions to examine results separately by age group. Future work should better represent the third and fourth decades of life and strive for balance across age distributions. Relatedly, this study was a secondary analysis of prior experiments, which contributed to differences across samples including versions of the go, no-go and stop-signal tasks, exclusion/inclusion criteria, and mood measures. Prospective studies would have better control of these parameters

although the present work demonstrated proof of concept for further investigations into neutral response inhibition, aging, and alexithymia.

Another potential limitation stemmed from only examining behavioral measures of inhibitory control. Previous work revealed that behavioral measures may be less sensitive to index the effects of alexithymia on inhibition and the no-go and stop-signal changes associated with aging (Hazlett Elverman, 2016; Nielson et al., 2002; Zhang et al., 2012). Thus, a future line of inquiry could incorporate these neural techniques.

A conservative approach was used in the exclusionary cutoffs for performance accuracy. Low motivation was suspected in one of the young adult samples, and as such, strict criteria for inclusion into the analyses were essential to assure internal validity. Participants were required to respond with 90% accuracy to target trials in no-go *and* stop, with 95% accuracy to go, *and* with 50% accuracy to inhibitory trials in no-go *and* stop. These performance criteria were applied across the entire dataset regardless of study or age group. Consequently, near perfect responding to targets was required at the outset of the experiment and had to be maintained throughout all three tasks. This precluded any variability in performance that could occur due to fatigue, and as such, participants with poorer sustained attention were likely to be excluded. This was potentially overly restrictive, particularly for older adults, as sustained attention abilities decline with age (Fortenbaugh et al., 2015). In fact, only 40% of the older adult sample remained after all exclusionary and inclusionary criteria were applied, yet 70% of the young adult sample were retained with the cutoffs. As such, participants, especially older individuals, who gave sufficient effort but did not respond near ceiling to targets on all three tasks were excluded. An alternative approach would be to analyze slightly different samples for each

task, allowing for better or poorer performance across the tasks. This approach would have somewhat different limitations as it would assume each task was conducted in isolation instead of maintaining the context of completing them in succession. Future work could also consider less stringent criteria. These alternative approaches would increase this study's external validity but may result in increased threats to internal validity. Furthermore, the shared variance among the mood composite, the TAS-20, and the subscores of the TAS-20 reduced the power of discernible effects of alexithymia and its factors on the predictive models thereby requiring multiple post hoc modeling. Multiple comparisons increase the likelihood of Type I Errors although the conservative cutoff approach reduced internal validity concerns. Given the lack of association between mood and most performance metrics, future directions for these data would identify targeted post hoc analyses to support the findings that alexithymia affects inhibitory control rather than psychiatric distress.

Conclusion

Across a large sample of individuals ranging in age from 18 to 92 years, older age predicted slower reaction times to target stimuli, especially among the middle-aged to older adult samples, which is consistent with prior work demonstrating relatively stable reaction time during early adulthood followed by progressive slowing in later life (Der & Deary, 2006). Age was generally not associated with accurate responding to target stimuli supporting the speed-accuracy tradeoff in later life for reaction time and inhibition tasks (Hazlett Elverman, 2016; Nielson et al., 2002; Williams et al., 1999). However, older age was associated with less accurate responding to inhibition trials of the stop-signal task and slowed suppression of motoric responses. Age did not predict no-go inhibition

performance. To some extent, these results replicate recent work demonstrating age-related deficits on both no-go and stop-signal tasks (Rey-Mermet & Gade, 2018).

Alexithymia, via the Difficulty Identifying Feelings (DIF) subscore, predicted greater commission errors on inhibition trials across both no-go and stop. Critically, mood symptoms did not moderate or mediate these relationships. These results reveal how information processing may be disrupted in alexithymia. That is, difficulties interpreting internal cues may lead to an inability to suppress competing responses and to selectively execute responses. Consistent with a processing theory of alexithymia, top-down and bottom-up information processing may be disrupted in high alexithymia (Frawley & Smith, 2001; Moriguchi & Komaki, 2013). Importantly, the effect of alexithymia on inhibitory control occurred in addition to aging effects suggesting that alexithymia may be a substantive contributor to age-related cognitive dysfunction (Correro II et al., 2019).

BIBLIOGRAPHY

Bagby, R. M., Parker, J. D., & Taylor, G. J. (1994). The twenty-item Toronto Alexithymia Scale--I. Item selection and cross-validation of the factor structure. *J Psychosom Res, 38*(1), 23-32.

Bagby, R. M., & Taylor, G. J. (1997). Measurement and validation of the alexithymia construct. In G. J. Taylor, R. M. Bagby, & J. D. A. Parker (Eds.), *Disorders of affect regulation: Alexithymia in medical and psychiatric illness* (pp. 46-66). Cambridge, UK: Cambridge University Press.

Bagby, R. M., Taylor, G. J., & Parker, J. D. (1994). The Twenty-item Toronto Alexithymia Scale--II. Convergent, discriminant, and concurrent validity. *J Psychosom Res, 38*(1), 33-40.

Bar-On, R., Tranel, D., Denburg, N. L., & Bechara, A. (2003). Exploring the neurological substrate of emotional and social intelligence. *Brain, 126*(Pt 8), 1790-1800. doi:10.1093/brain/awg177

Barsky, A. J., Goodson, J. D., Lane, R. S., & Cleary, P. D. (1988). The amplification of somatic symptoms. *Psychosom Med, 50*(5), 510-519.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: psychometric properties. *J Consult Clin Psychol, 56*(6), 893-897. doi:10.1037//0022-006x.56.6.893

Beck, A. T., & Steer, R. A. (1990). Manual for the Beck Anxiety Inventory. *San Antonio, TX: Psychological Corporation*.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Beck Depression Inventory-II (BDI-II). *San Antonio, TX: Psychological Corporation*.

Berg, E. A. (1948). A simple objective technique for measuring flexibility in thinking. *J Gen Psychol, 39*, 15-22. doi:10.1080/00221309.1948.9918159

Blair, J. R., & Spreen, O. (1989). Predicting premorbid IQ: A revision of the national adult reading test. *The Clinical Neuropsychologist, 3*(2), 129-136. doi:10.1080/13854048908403285

Bucci, W. (2001). Pathways of emotional communication. *Psychoanalytic Inquiry, 21*(1), 40-70. doi:10.1080/07351692109348923

Cabeza, R. (2002). Hemispheric asymmetry reduction in older adults: the HAROLD model. *Psychol Aging, 17*(1), 85-100. doi:10.1037//0882-7974.17.1.85

Carstensen, L. L., & Mikels, J. A. (2005). At the intersection of emotion and cognition: Aging and the positivity effect. *Curr Dir Psychol Sci, 14*(3), 117-121. doi:10.1111/j.0963-7214.2005.00348.x

Caserta, M. T., Bannon, Y., Fernandez, F., Giunta, B., Schoenberg, M. R., & Tan, J. (2009). Normal brain aging clinical, immunological, neuropsychological, and neuroimaging features. *Int Rev Neurobiol, 84*, 1-19. doi:10.1016/S0074-7742(09)00401-2

Charles, S. T., Mather, M., & Carstensen, L. L. (2003). Aging and emotional memory: the forgettable nature of negative images for older adults. *J Exp Psychol Gen, 132*(2), 310-324.

Charlot, V., & Feyereisen, P. (2004). Aging and the deletion function of inhibition. *Aging, Neuropsychology, and Cognition, 11*(1), 12-24. doi:10.1076/anec.11.1.12.29363

Congdon, E., Mumford, J. A., Cohen, J. R., Galvan, A., Canli, T., & Poldrack, R. A. (2012). Measurement and reliability of response inhibition. *Front Psychol, 3*, 37. doi:10.3389/fpsyg.2012.00037

Correro II, A. N., Paitel, E. R., Byers, S. J., & Nielson, K. A. (2019). The role of alexithymia in memory and executive functioning across the lifespan. *Cogn Emot*, 1-16. doi:10.1080/02699931.2019.1659232

Darowski, E. S., Helder, E., Zacks, R. T., Hasher, L., & Hambrick, D. Z. (2008). Age-related differences in cognition: the role of distraction control. *Neuropsychology, 22*(5), 638-644. doi:10.1037/0894-4105.22.5.638

Der, G., & Deary, I. J. (2006). Age and sex differences in reaction time in adulthood: results from the United Kingdom Health and Lifestyle Survey. *Psychol Aging, 21*(1), 62-73. doi:10.1037/0882-7974.21.1.62

Donders, F. C. (1969). On the speed of mental processes. *Acta Psychol (Amst), 30*, 412-431.

Dressaire, D., Stone, C. B., Nielson, K. A., Guerdoux, E., Martin, S., Brouillet, D., & Luminet, O. (2015). Alexithymia impairs the cognitive control of negative material while facilitating the recall of neutral material in both younger and older adults. *Cogn Emot, 29*(3), 442-459. doi:10.1080/02699931.2014.919898

Eastabrook, J. M., Lanteigne, D. M., & Hollenstein, T. (2013). Decoupling between physiological, self-reported, and expressed emotional responses in alexithymia. *Pers Indiv Differ, 55*(8), 978-982. doi:10.1016/j.paid.2013.08.001

Egner, T., Etkin, A., Gale, S., & Hirsch, J. (2008). Dissociable neural systems resolve conflict from emotional versus nonemotional distracters. *Cereb Cortex, 18*(6), 1475-1484. doi:10.1093/cercor/bhm179

Elliott, R. (2003). Executive functions and their disorders. *Br Med Bull, 65*, 49-59.

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics, 16*(1), 143-149. doi:10.3758/BF03203267

Faul, F., Erdfelder, E., Lang, A. G., & Buchner, A. (2007). G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods, 39*(2), 175-191.

Fjell, A. M., Westlye, L. T., Amlien, I., Espeseth, T., Reinvang, I., Raz, N., . . . Walhovd, K. B. (2009). High consistency of regional cortical thinning in aging across multiple samples. *Cereb Cortex, 19*(9), 2001-2012. doi:10.1093/cercor/bhn232

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini-mental state": a practical method for grading the cognitive state of patients for the clinician. *Journal of psychiatric research, 12*(3), 189-198.

Fortenbaugh, F. C., DeGutis, J., Germine, L., Wilmer, J. B., Grosso, M., Russo, K., & Esterman, M. (2015). Sustained Attention Across the Life Span in a Sample of 10,000: Dissociating Ability and Strategy. *Psychol Sci, 26*(9), 1497-1510. doi:10.1177/0956797615594896

Frawley, W., & Smith, R. N. (2001). A processing theory of alexithymia. *Cognitive Systems Research, 2*(3), 189-206. doi:10.1016/S1389-0417(01)00029-8

Friedman, N. P., Miyake, A., Altamirano, L. J., Corley, R. P., Young, S. E., Rhea, S. A., & Hewitt, J. K. (2016). Stability and change in executive function abilities from late adolescence to early adulthood: A longitudinal twin study. *Dev Psychol, 52*(2), 326-340. doi:10.1037/dev0000075

Fydrich, T., Dowdall, D., & Chambless, D. L. (1992). Reliability and validity of the Beck Anxiety Inventory. *Journal of Anxiety Disorders, 6*(1), 55-61. doi:10.1016/0887-6185(92)90026-4

Garavan, H., Ross, T. J., & Stein, E. A. (1999). Right hemispheric dominance of inhibitory control: an event-related functional MRI study. *Proc Natl Acad Sci U S A, 96*(14), 8301-8306. doi:10.1073/pnas.96.14.8301

Goerlich, K. S. (2018). Electrophysiology of alexithymia. In O. Luminet, R. M. Bagby, & G. J. Taylor (Eds.), *Alexithymia: Advances in research, theory, and clinical practice* (pp. 250-266). Cambridge, United Kingdom: Cambridge University Press.

Goerlich, K. S., & Aleman, A. (2018). Neuroimaging studies of alexithymia. In O. Luminet, R. M. Bagby, & G. J. Taylor (Eds.), *Alexithymia: Advances in research, theory, and clinical practice* (pp. 207-249). Cambridge, United Kingdom: Cambridge University Press.

Goh, J. O., & Park, D. C. (2009). Neuroplasticity and cognitive aging: the scaffolding theory of aging and cognition. *Restor Neurol Neurosci, 27*(5), 391-403. doi:10.3233/RNN-2009-0493

Grant, D. A., & Berg, E. A. (1948). A behavioral analysis of degree of reinforcement and ease of shifting to new responses in a Weigl-type card-sorting problem. *J Exp Psychol, 38*(4), 404-411.

Gunzelmann, T., Kupfer, J., & Brahler, E. (2002). Alexithymia in the elderly general population. *Compr Psychiatry, 43*(1), 74-80.

Hamm, V. P., & Hasher, L. (1992). Age and the availability of inferences. *Psychol Aging, 7*(1), 56-64.

Hasher, L., & Zacks, R. T. (1988). Working memory, comprehension, and aging: A review and a new view. *Psychology of Learning and Motivation, 22*, 193-225. doi:10.1016/S0079-7421(08)60041-9

Hashtroudi, S., Johnson, M. K., & Chrosniak, L. D. (1990). Aging and qualitative characteristics of memories for perceived and imagined complex events. *Psychol Aging, 5*(1), 119-126.

Haviland, M. G., & Reise, S. P. (1996). A California Q-set alexithymia prototype and its relationship to ego-control and ego-resiliency. *J Psychosom Res, 41*(6), 597-607.

Hayes, A. F. (2018). *Introduction to mediation, moderation, and conditional process analysis : a regression-based approach* (Second edition. ed.). New York: Guilford Press.

Hazlett Elverman, K. (2016). *A behavioral and neural investigation of the impact of age and genetic risk for Alzheimer's disease on inhibitory control.* Marquette University, e-Publications@Marquette.

Head, D., Kennedy, K. M., Rodrigue, K. M., & Raz, N. (2009). Age differences in perseveration: cognitive and neuroanatomical mediators of performance on the Wisconsin Card Sorting Test. *Neuropsychologia, 47*(4), 1200-1203. doi:10.1016/j.neuropsychologia.2009.01.003

Henry, J. D., Phillips, L. H., Crawford, J. R., Theodorou, G., & Summers, F. (2006). Cognitive and psychosocial correlates of alexithymia following traumatic brain injury. *Neuropsychologia, 44*(1), 62-72. doi:10.1016/j.neuropsychologia.2005.04.011

Hofmann, W., Schmeichel, B. J., & Baddeley, A. D. (2012). Executive functions and self-regulation. *Trends in Cognitive Sciences, 16*(3), 174-180. doi:10.1016/j.tics.2012.01.006

Honkalampi, K., De Berardis, D., Vellante, F., & Viinamaki, H. (2018). Relations between alexithymia and depressive and anxiety disorders and personality. In O. Luminet, R. M. Bagby, & G. J. Taylor (Eds.), *Alexithymia: Advances in research, theory, and clinical practice* (pp. 142-157). Cambridge, United Kingdom: Cambridge University Press.

Honkalampi, K., Koivumaa-Honkanen, H., Lehto, S. M., Hintikka, J., Haatainen, K., Rissanen, T., & Viinamaki, H. (2010). Is alexithymia a risk factor for major depression, personality disorder, or alcohol use disorders? A prospective population-based study. *J Psychosom Res, 68*(3), 269-273. doi:10.1016/j.jpsychores.2009.05.010

Inslegers, R., Vanheule, S., Meganck, R., Debaere, V., Trenson, E., & Desmet, M. (2012). Interpersonal problems and cognitive characteristics of interpersonal representations in alexithymia: a study using a self-report and interview-based measure of alexithymia. *J Nerv Ment Dis, 200*(7), 607-613. doi:10.1097/NMD.0b013e31825bfad9

Jurica, S. J., Leitten, C. L., & Mattis, S. (2001). Dementia Rating Scale: Professional manual. In. Odessa, FL: Psychological Assessment Resources.

Kano, M., & Fukudo, S. (2013). The alexithymic brain: the neural pathways linking alexithymia to physical disorders. *Biopsychosoc Med, 7*(1), 1. doi:10.1186/1751-0759-7-1

Kano, M., Hamaguchi, T., Itoh, M., Yanai, K., & Fukudo, S. (2007). Correlation between alexithymia and hypersensitivity to visceral stimulation in human. *Pain, 132*(3), 252-263. doi:10.1016/j.pain.2007.01.032

Kausler, D. H., & Hakami, M. K. (1982). Frequency judgments by young and elderly adults for relevant stimuli with simultaneously present irrelevant stimuli. *J Gerontol, 37*(4), 438-442.

Keefer, K. V., Taylor, G. J., Parker, J. D. A., & Bagby, R. M. (2019). Taxometric Analysis of the Toronto Structured Interview for Alexithymia: Further Evidence That Alexithymia Is a Dimensional Construct. *Assessment, 26*(3), 364-374. doi:10.1177/1073191117698220

Koven, N. S., & Thomas, W. (2010). Mapping facets of alexithymia to executive dysfunction in daily life. *Pers Indiv Differ, 49*, 24-28. doi:10.1016/j.paid.2010.02.034

Lamberty, G. J., & Holt, C. S. (1995). Evidence for a verbal deficit in alexithymia. *J Neuropsychiatry Clin Neurosci, 7*(3), 320-324. doi:10.1176/jnp.7.3.320

Lane, R. D., Ahern, G. L., Schwartz, G. E., & Kaszniak, A. W. (1997). Is alexithymia the emotional equivalent of blindsight? *Biol Psychiatry, 42*(9), 834-844.

Lane, R. D., & Schwartz, G. E. (1987). Levels of emotional awareness: a cognitive-developmental theory and its application to psychopathology. *Am J Psychiatry, 144*(2), 133-143. doi:10.1176/ajp.144.2.133

Lane, R. D., Sechrest, L., & Riedel, R. (1998). Sociodemographic correlates of alexithymia. *Compr Psychiatry, 39*(6), 377-385.

Langenecker, S. A., Zubieta, J. K., Young, E. A., Akil, H., & Nielson, K. A. (2007). A task to manipulate attentional load, set-shifting, and inhibitory control: convergent validity and test-retest reliability of the Parametric Go/No-Go Test. *J Clin Exp Neuropsychol, 29*(8), 842-853. doi:10.1080/13803390601147611

Li, S., Zhang, B., Guo, Y., & Zhang, J. (2015). The association between alexithymia as assessed by the 20-item Toronto Alexithymia Scale and depression: A meta-analysis. *Psychiatry Res, 227*(1), 1-9. doi:10.1016/j.psychres.2015.02.006

Lipszyc, J., & Schachar, R. (2010). Inhibitory control and psychopathology: a meta-analysis of studies using the stop signal task. *J Int Neuropsychol Soc, 16*(6), 1064-1076. doi:10.1017/S1355617710000895

Logan, G. D., & Cowan, W. B. (1984). On the ability to inhibit thought and action: A theory of an act of control. *Psychol Rev, 91*(3), 295-327. doi:10.1037/0033-295X.91.3.295

Logan, G. D., Cowan, W. B., & Davis, K. A. (1984). On the ability to inhibit simple and choice reaction time responses: a model and a method. *J Exp Psychol Hum Percept Perform, 10*(2), 276-291.

Luminet, O., Vermeulen, N., Demaret, C., Taylor, G. J., & Bagby, R. M. (2006). Alexithymia and levels of processing: Evidence for an overall deficit in remembering emotion words. *J Res Pers, 40*(5), 713-733. doi:10.1016/j.jrp.2005.09.001

Luminet, O., & Zamariola, G. (2018). Emotion knowledge and emotion regulation in alexithymia. In O. Luminet, R. M. Bagby, & G. J. Taylor (Eds.), *Alexithymia: Advances in research, theory, and clinical practice* (pp. 49-77). Cambridge, United Kingdom: Cambridge University Press.

Lumley, M. A. (2000). Alexithymia and negative emotional conditions. *J Psychosom Res, 49*(1), 51-54. doi:10.1016/s0022-3999(00)00161-6

Lustig, C., Hasher, L., & Zacks, R. (2007). Inhibitory deficit theory: Recent developments in a "new view". In D. S. Gorfein & C. M. MacLeod (Eds.), *Inhibition in Cognition*. Washington, D.C.: American Psychological Association.

Lyche, P., Jonassen, R., Stiles, T. C., Ulleberg, P., & Landro, N. I. (2010). Cognitive Control Functions in Unipolar Major Depression with and without Co-Morbid Anxiety Disorder. *Front Psychiatry, 1*, 149. doi:10.3389/fpsyt.2010.00149

MacCormack, J. K., & Lindquist, K. A. (2018). Feeling hangry? When hunger is conceptualized as emotion. *Emotion*. doi:10.1037/emo0000422

Marchesi, C., Brusamonti, E., & Maggini, C. (2000). Are alexithymia, depression, and anxiety distinct constructs in affective disorders? *J Psychosom Res, 49*(1), 43-49. doi:10.1016/s0022-3999(00)00084-2

Marchesi, C., Ossola, P., Tonna, M., & De Panfilis, C. (2014). The TAS-20 more likely measures negative affects rather than alexithymia itself in patients with major depression, panic disorder, eating disorders and substance use disorders. *Compr Psychiatry, 55*(4), 972-978. doi:10.1016/j.comppsych.2013.12.008

Mather, M. (2012). The emotion paradox in the aging brain. *Ann N Y Acad Sci, 1251*, 33-49. doi:10.1111/j.1749-6632.2012.06471.x

Mather, M., & Carstensen, L. L. (2005). Aging and motivated cognition: the positivity effect in attention and memory. *Trends Cogn Sci, 9*(10), 496-502. doi:10.1016/j.tics.2005.08.005

Mather, M., & Knight, M. (2005). Goal-directed memory: the role of cognitive control in older adults' emotional memory. *Psychol Aging, 20*(4), 554-570. doi:10.1037/0882-7974.20.4.554

Mattila, A. K., Keefer, K. V., Taylor, G. J., Joukamaa, M., Jula, A., Parker, J. D. A., & Bagby, R. M. (2010). Taxometric analysis of alexithymia in a general population sample from Finland. *Pers Indiv Differ, 49*(3), 216-221. doi:10.1016/j.paid.2010.03.038

Mattila, A. K., Salminen, J. K., Nummi, T., & Joukamaa, M. (2006). Age is strongly associated with alexithymia in the general population. *J Psychosom Res, 61*(5), 629-635. doi:10.1016/j.jpsychores.2006.04.013

Mattis, S. (1988). Dementia Rating Scale Professional Manual. In. Odessa, FL: Psychological Assessment Resources.

Meltzer, M. A., & Nielson, K. A. (2010). Memory for emotionally provocative words in alexithymia: a role for stimulus relevance. *Conscious Cogn, 19*(4), 1062-1068. doi:10.1016/j.concog.2010.05.008

Messina, A., Beadle, J. N., & Paradiso, S. (2014). Towards a classification of alexithymia: Primary, secondary and organic. *Journal of Psychopathology, 20*(1), 38-49.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "Frontal Lobe" tasks: a latent variable analysis. *Cogn Psychol, 41*(1), 49-100. doi:10.1006/cogp.1999.0734

Monsch, A. U., Bondi, M. W., Salmon, D. P., Butters, N., Thal, L. J., Hansen, L. A., . . . Klauber, M. R. (1995). Clinical validity of the Mattis Dementia Rating Scale in detecting Dementia of the Alzheimer type. A double cross-validation and application to a community-dwelling sample. *Arch Neurol, 52*(9), 899-904. doi:10.1001/archneur.1995.00540330081018

Moriguchi, Y., & Komaki, G. (2013). Neuroimaging studies of alexithymia: physical, affective, and social perspectives. *Biopsychosoc Med, 7*(1), 8. doi:10.1186/1751-0759-7-8

Murphy, J., Catmur, C., & Bird, G. (2017). Alexithymia Is Associated With a Multidomain, Multidimensional Failure of Interoception: Evidence From Novel Tests. *J Exp Psychol Gen*. doi:10.1037/xge0000366

Nemiah, J. C. (1977). Alexithymia. Theoretical considerations. *Psychother Psychosom, 28*(1-4), 199-206. doi:10.1159/000287064

Nemiah, J. C., Freyberger, H., & Sifneos, P. E. (1976). Alexithymia: A view of the psychosomatic process. In O. W. Hill (Ed.), *Modern Trends in Psychosomatic Medicine* (Vol. 3, pp. 430-439). London, UK: Butterworths.

Nielson, K. A., Langenecker, S. A., & Garavan, H. (2002). Differences in the functional neuroanatomy of inhibitory control across the adult life span. *Psychol Aging, 17*(1), 56-71.

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: views from cognitive and personality psychology and a working inhibition taxonomy. *Psychol Bull, 126*(2), 220-246.

Onor, M., Trevisiol, M., Spano, M., Aguglia, E., & Paradiso, S. (2010). Alexithymia and aging: a neuropsychological perspective. *J Nerv Ment Dis, 198*(12), 891-895. doi:10.1097/NMD.0b013e3181fe743e

Papciak, A. S., Feuerstein, M., & Spiegel, J. A. (1985). Stress reactivity in alexithymia: decoupling of physiological and cognitive responses. *J Human Stress, 11*(3), 135-142. doi:10.1080/0097840X.1985.9936750

Paradiso, S., Vaidya, J. G., McCormick, L. M., Jones, A., & Robinson, R. G. (2008). Aging and alexithymia: association with reduced right rostral cingulate volume. *Am J Geriatr Psychiatry, 16*(9), 760-769. doi:10.1097/JGP.0b013e31817e73b0

Park, D. C., & Reuter-Lorenz, P. (2009). The adaptive brain: aging and neurocognitive scaffolding. *Annual Review of Psychology, Vol 64, 60*, 173-196. doi:10.1146/annurev.psych.59.103006.093656

Park, D. C., Smith, A. D., Lautenschlager, G., Earles, J. L., Frieske, D., Zwahr, M., & Gaines, C. L. (1996). Mediators of long-term memory performance across the life span. *Psychol Aging, 11*(4), 621-637.

Parker, J. D., Bagby, R. M., & Taylor, G. J. (1991). Alexithymia and depression: distinct or overlapping constructs? *Compr Psychiatry, 32*(5), 387-394. doi:10.1016/0010-440x(91)90015-5

Parker, J. D., Keefer, K. V., Taylor, G. J., & Bagby, R. M. (2008). Latent structure of the alexithymia construct: a taxometric investigation. *Psychol Assess, 20*(4), 385-396. doi:10.1037/a0014262

Parker, J. D., Taylor, G. J., & Bagby, R. M. (2003). The 20-Item Toronto Alexithymia Scale. III. Reliability and factorial validity in a community population. *J Psychosom Res, 55*(3), 269-275.

Pasini, A., Delle Chiaie, R., Seripa, S., & Ciani, N. (1992). Alexithymia as related to sex, age, and educational level: results of the Toronto Alexithymia Scale in 417 normal subjects. *Compr Psychiatry, 33*(1), 42-46.

Preece, D., Becerra, R., Allan, A., Robinson, K., & Dandy, J. (2017). Establishing the theoretical components of alexithymia via factor analysis: Introduction and validation of the attention-appraisal model of alexithymia. *Personality and Individual Differences, 119*, 341-352. doi:10.1016/j.paid.2017.08.003

Preece, D., Becerra, R., Robinson, K., & Dandy, J. (2018). Assessing alexithymia: Psychometric properties and factorial invariance of the 20-item Toronto Alexithymia Scale in nonclinical and psychiatric samples. *J Psychopathol Behav Assess, 40*(2), 276-287. doi:10.1007/s10862-017-9634-6

Reuter-Lorenz, P. A., & Park, D. C. (2014). How does it STAC up? Revisiting the scaffolding theory of aging and cognition. *Neuropsychol Rev, 24*(3), 355-370. doi:10.1007/s11065-014-9270-9

Rey-Mermet, A., & Gade, M. (2018). Inhibition in aging: What is preserved? What declines? A meta-analysis. *Psychon Bull Rev, 25*(5), 1695-1716. doi:10.3758/s13423-017-1384-7

Ricciardi, L., Demartini, B., Fotopoulou, A., & Edwards, M. J. (2015). Alexithymia in Neurological Disease: A Review. *J Neuropsychiatry Clin Neurosci, 27*(3), 179-187. doi:10.1176/appi.neuropsych.14070169

Rinaldi, R., Radian, V., Rossignol, M., Arachchige, K. G. K., & Lefebvre, L. (2017). Thinking About One's Feelings Association Between Alexithymia and Cognitive Styles in a Nonclinical Population. *Journal of Nervous and Mental Disease, 205*(10), 812-815. doi:10.1097/Nmd.0000000000000721

Rubia, K., Russell, T., Overmeyer, S., Brammer, M. J., Bullmore, E. T., Sharma, T., . . . Taylor, E. (2001). Mapping motor inhibition: conjunctive brain activations across different versions of go/no-go and stop tasks. *Neuroimage, 13*(2), 250-261. doi:10.1006/nimg.2000.0685

Ryder, A. G., Sunohara, M., Dere, J., & Chentsova-Dutton, Y. E. (2018). The cultural shaping of alexithymia. In O. Luminet, R. M. Bagby, & G. J. Taylor (Eds.), *Alexithymia: Advances in research, theory, and clinical practice* (pp. 33-48). Cambridge, United Kingdom: Cambridge University Press.

Salminen, J. K., Saarijarvi, S., Aarela, E., Toikka, T., & Kauhanen, J. (1999). Prevalence of alexithymia and its association with sociodemographic variables in the general population of Finland. *J Psychosom Res, 46*(1), 75-82.

Salthouse, T. A. (2010). Selective review of cognitive aging. *J Int Neuropsychol Soc, 16*(5), 754-760. doi:10.1017/S1355617710000706

Santorelli, G. D., & Ready, R. E. (2015). Alexithymia and Executive Function in Younger and Older Adults. *Clin Neuropsychol, 29*(7), 938-955. doi:10.1080/13854046.2015.1123296

Sekely, A., Bagby, R. M., & Porcelli, P. (2018). Assessment of the alexithymia construct. In O. Luminet, R. M. Bagby, & G. J. Taylor (Eds.), *Alexithymia: Advances in research, theory, and clinical practice* (pp. 17-32). Cambridge, United Kingdom: Cambridge University Press.

Sifneos, P. E. (1973). The prevalence of 'alexithymic' characteristics in psychosomatic patients. *Psychother Psychosom, 22*(2), 255-262.

Strauss, E., Sherman, E. M. S., Spreen, O., & Spreen, O. (2006). *A compendium of neuropsychological tests : administration, norms, and commentary* (3rd ed.). Oxford ; New York: Oxford University Press.

Sturm, V. E., & Levenson, R. W. (2011). Alexithymia in neurodegenerative disease. *Neurocase, 17*(3), 242-250. doi:10.1080/13554794.2010.532503

Taylor, G. J. (2000). Recent developments in alexithymia theory and research. *Can J Psychiatry, 45*(2), 134-142. doi:10.1177/070674370004500203

Taylor, G. J., & Bagby, R. M. (2004). New trends in alexithymia research. *Psychother Psychosom, 73*(2), 68-77. doi:10.1159/000075537

Tombaugh, T. N., & McIntyre, N. J. (1992). The mini-mental state examination: a comprehensive review. *J Am Geriatr Soc, 40*(9), 922-935. doi:10.1111/j.1532-5415.1992.tb01992.x

Vaidya, J. G., Paradiso, S., Boles Ponto, L. L., McCormick, L. M., & Robinson, R. G. (2007). Aging, grey matter, and blood flow in the anterior cingulate cortex. *Neuroimage, 37*(4), 1346-1353. doi:10.1016/j.neuroimage.2007.06.015

Verbruggen, F., & Logan, G. D. (2008). Automatic and controlled response inhibition: associative learning in the go/no-go and stop-signal paradigms. *J Exp Psychol Gen, 137*(4), 649-672. doi:10.1037/a0013170

Vermeulen, N., Domachowska, I., & Nielson, K. A. (2018). Memory and executive functions in alexithymia. In O. Luminet, R. M. Bagby, & G. J. Taylor (Eds.), *Alexithymia: Advances in research, theory, and clinical practice* (pp. 78-89). Cambridge, United Kingdom: Cambridge University Press.

Vermeulen, N., & Luminet, O. (2009). Alexithymia factors and memory performances for neutral and emotional words. *Pers Indiv Differ, 47*, 305-309. doi:10.1016/j.paid.2009.03.018

Votruba, K. L., & Langenecker, S. A. (2013). Factor structure, construct validity, and age- and education-based normative data for the Parametric Go/No-Go Test. *J Clin Exp Neuropsychol, 35*(2), 132-146. doi:10.1080/13803395.2012.758239

Votruba, K. L., Rapport, L. J., Vangel, S. J., Jr., Hanks, R. A., Lequerica, A., Whitman, R. D., & Langenecker, S. (2008). Impulsivity and traumatic brain injury: the relations among behavioral observation, performance measures, and rating scales. *J Head Trauma Rehabil, 23*(2), 65-73. doi:10.1097/01.HTR.0000314525.93381.69

Wastell, C. A., & Taylor, A. J. (2002). Alexithymic mentalising: Theory of mind and social adaptation. *Social Behavior and Personality, 30*(2), 141-148. doi:10.2224/sbp.2002.30.2.141

Whalen, P. J., Bush, G., McNally, R. J., Wilhelm, S., McInerney, S. C., Jenike, M. A., & Rauch, S. L. (1998). The emotional counting Stroop paradigm: a functional magnetic resonance imaging probe of the anterior cingulate affective division. *Biol Psychiatry, 44*(12), 1219-1228.

Williams, B. R., Ponesse, J. S., Schachar, R. J., Logan, G. D., & Tannock, R. (1999). Development of inhibitory control across the life span. *Dev Psychol, 35*(1), 205-213. doi:10.1037//0012-1649.35.1.205

Wingfield, A., Stine, E. A., Lahar, C. J., & Aberdeen, J. S. (1988). Does the capacity of working memory change with age? *Exp Aging Res, 14*(2-3), 103-107. doi:10.1080/03610738808259731

Wood, R. L., & Williams, C. (2007). Neuropsychological correlates of organic alexithymia. *J Int Neuropsychol Soc, 13*(3), 471-479. doi:10.1017/S1355617707070518

Wright, L., Lipszyc, J., Dupuis, A., Thayapararajah, S. W., & Schachar, R. (2014). Response inhibition and psychopathology: a meta-analysis of go/no-go task performance. *J Abnorm Psychol, 123*(2), 429-439. doi:10.1037/a0036295

Yesavage, J. A., Brink, T. L., Rose, T. L., Lum, O., Huang, V., Adey, M., & Leirer, V. O. (1982). Development and validation of a geriatric depression screening scale: a preliminary report. *Journal of psychiatric research, 17*(1), 37-49.

Yuruyen, M., Akcan, F. E., Batun, G. C., Gultekin, G., Toprak, M., Yavuzer, H., & Emul, M. (2017). Alexithymia in people with subjective cognitive decline, mild cognitive impairment, and mild Alzheimer's disease. *Aging Clin Exp Res*. doi:10.1007/s40520-017-0725-8

Zelazo, P. D., & Cunningham, W. (2007). Executive function: Mechanisms underlying emotion regulation. In J. Gross (Ed.), *Handbook of emotion regulation* (pp. 135-158). New York, NY: Guilford.

Zhang, L., Ye, R., Yu, F., Cao, Z., Zhu, C., Cai, Z., . . . Wang, K. (2012). How does emotional context modulate response inhibition in alexithymia: electrophysiological evidence from an ERP study. *PLoS One, 7*(12), e51110. doi:10.1371/journal.pone.0051110

Zhang, L., Zhu, C., Ye, R., Cao, Z., Tian, Y., Yang, P., . . . Wang, K. (2011). Impairment of conflict processing in alexithymic individuals. *Neurosci Lett, 504*(3), 261-264. doi:10.1016/j.neulet.2011.09.043

Zhu, X. Z., Wang, X. Y., Huang, Y., Yao, S. Q., & Tang, H. B. (2006). A comparative study of Wisconsin Card Sorting Test in individuals with different degrees of alexithymia. *Chinese Journal of Clinical Psychology, 14*(2), 132-133.